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By AMEDEE GRANGER, K.C.B., K.C.I., M.D., F.A.C.R.,

Professor of Radiology, Louisiana State University, Medical Center; Director of the Department of Radiology, Louisiana State Charity Hospital, New Orleans.

186 Pages, 113 Engravings. Price $5.50

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Attend Third Annual Assembly of the Southeastern Surgical Congress, Birmingham, Alabama, March 7-8, 1932. Tutwiler Hotel.

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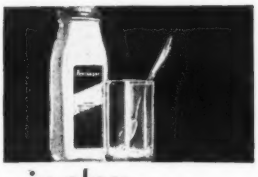

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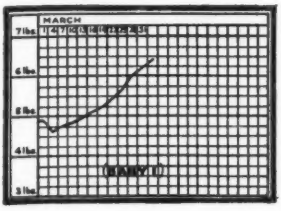


These charts have been taken from the hospital records of the prematures born in this hospital* between December 15, 1929, and March 1, 1930, and fed with Similac, the scientific milk modification for infants deprived of breast milk.

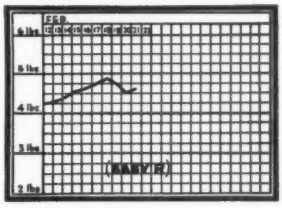
* Name of hospital on request.

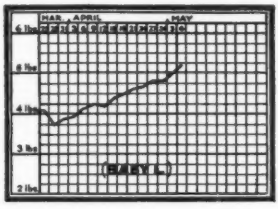
BABY I. Born March 1, 1930

Birth Day	Weight 4 pounds, 14 ounces	Feeding History	No. Feedings in 24 hrs.
Mar. 1-30	Similac, 1 ounce,	every 2 hrs. Fed with Breck Feeder	12
Mar. 2-30	Similac, 1 ounce,	every 2 hrs. Fed with Breck Feeder	12
Mar. 6-30	Similac, 1 ounce,	every 2 hrs. Fed with nursing bottle	12
Mar. 7-30	Similac, 2 ounces,	every 2 hrs. Fed with nursing bottle	8
Mar. 31-30	Similac, 3 ounces,	every 3 hrs. Fed with nursing bottle	8
Mar. 31-30	Discharged from Hospital		

BABY R. Born Feb. 12th, 1930

Birth Day	Weight 4 pounds, 4 ounces	Feeding History	No. Feedings in 24 hrs.
Feb. 12-30	Similac,	½ drahm every 2 hrs. Fed with medicine dropper	12
Feb. 13-30	Similac,	1 drahm every 2 hrs. Fed with medicine dropper	12
Feb. 14-30	Similac,	1½ ounces every 2 hrs. Fed with Breck Feeder	12
Feb. 15-30	Similac,	2 ounces every 2 hrs. Fed with Breck Feeder	12
Feb. 16-30	Similac,	2 ounces every 2 hrs. Fed with Breck Feeder	12
Feb. 19-30	—Breast—every 2 hrs. Complemented with 1 oz. Similac		12
Feb. 21-30	Breast feeding discontinued. Similac 2 ounces every 2 hrs. Fed with nursing bottle		12
Feb. 22-30	Similac, 2 ounces every 2 hrs. from nursing bottle		12
Feb. 22-30	Discharged from Hospital.		



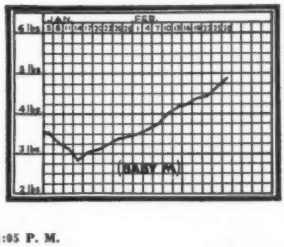


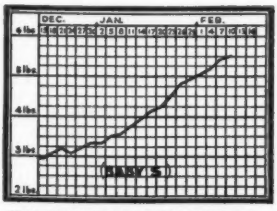
BABY L. Born March 25, 1930

Weight at birth, 4 pounds and 1 ounce					No. Feedings in 24 hrs.
Day	Feeding History				
Mar. 25-30	Water,	every 2 hrs.	Fed with	bottle	12
Mar. 26-30	Similac,	1 drahm	every 2 hrs.	Fed with bottle	12
Mar. 27-30	Similac,	1 drahm	every 2 hrs.	Fed with bottle	12
Mar. 28-30	Similac,	2 drahms	every 2 hrs.	Fed with bottle	12
Mar. 29-30	Similac,	½ ounce	every 2 hrs.	Fed with bottle	12
Mar. 31-30	Similac,	1 ounce	every 2 hrs.	Fed with bottle	12
Apr. 11-30	Similac,	1½ ounces	every 2 hrs.	Fed with bottle	12
Apr. 15-30	Similac,	2 ounces	every 2 hrs.	Fed with bottle	12
May 1-30	Similac,	2½ ounces	every 2 hrs.	Fed with bottle	12
May 6-30	Similac,	2½ ounces	every 2 hrs.	Fed with bottle	12
May 6-30	Discharged from Hospital.				

BABY M. Born Jan. 5, 1930 at 11:25 A. M.

Birth Day	Weight 3 pounds, 9 ounces	Feeding History	No. Feedings in 24 hrs.
Jan. 5-30	Similac,	2 drahms every 2 hrs. Fed with medicine dropper	12
Jan. 6-30	Similac,	2 drahms every 2 hrs. Fed with medicine dropper	12
Jan. 7-30	Similac,	½ ounce every 2 hrs. Fed with medicine dropper	12
Jan. 10-30	Breast Feeding, every 2 hrs.		12
Jan. 16-30	Breast discontinued. Similac, (concentrated double strength). 1 ounce every 2 hrs. Fed by Gavage		12
Jan. 19-30	Similac, concentrated, 1 ounce every 2 hrs. Fed with Breck Feeder		10
Jan. 22-30	Similac, standard dilution, 1½ ounces every 2 hrs. Fed with nursing bottle		10
Jan. 31-30	Similac, 2 ounces	every 2 hrs. Fed with nursing bottle	10
Feb. 10-30	Similac, 2½ ounces	every 2 hrs. Fed with nursing bottle	8
Feb. 28-30	Similac, 3 ounces	every 2 hrs. Fed with nursing bottle	8
Feb. 28-30	Discharged from Hospital.		





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Dec. 16-29	Similac,	1 drahm every 2 hrs. Fed with medicine dropper	12
Dec. 17-29	Similac,	2 drahms every 2 hrs. Fed with medicine dropper	12
Dec. 18-29	Similac,	3 drahms every 2 hrs. Fed with medicine dropper	12
Dec. 19-29	Similac,	4 drahms every 2 hrs. Fed with medicine dropper	12
Dec. 20-29	Similac,	4 drahms every 2 hrs. Fed with medicine dropper	12
Dec. 21-29	Similac,	4 drahms every 2 hrs. Fed with medicine dropper	12
Dec. 22-29	Similac,	4 drahms every 2 hrs. Fed with medicine dropper	12
Dec. 23-29	Similac,	5 drahms every 2 hrs. Fed with medicine dropper	12
Dec. 24-29	Similac,	5 drahms every 2 hrs. Fed with medicine dropper	12
Dec. 25-29	Similac,	6 drahms every 2 hrs. Fed with medicine dropper	12
Dec. 26-29	Similac,	6 drahms every 2 hrs. Fed with Breck Feeder	12
Dec. 27-29	Similac,	1 ounce every 2 hrs. Fed with Breck Feeder	12
Jan. 8-30	Similac,	1 ounce every 2 hrs. Fed with nursing bottle	12
Jan. 11-30	Similac,	1 ounce every 2 hrs. Fed with nursing bottle	12
Jan. 13-30	Similac,	1½ ounces every 2 hrs. Fed with nursing bottle	10
Jan. 17-30	Similac,	2 ounces every 2 hrs. Fed with nursing bottle	10
Jan. 23-30	Similac,	2½ ounces every 2 hrs. Fed with nursing bottle	10
Jan. 27-30	Similac,	3 ounces every 2 hrs. Fed with nursing bottle	8
Feb. 2-30	Similac,	3 ounces every 3 hrs. Fed with nursing bottle	8
Feb. 10-30	Similac,	3 ounces every 3 hrs. Fed with nursing bottle	8
Feb. 10-30	Discharged from Hospital.		

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Potent in the presence of organic matter

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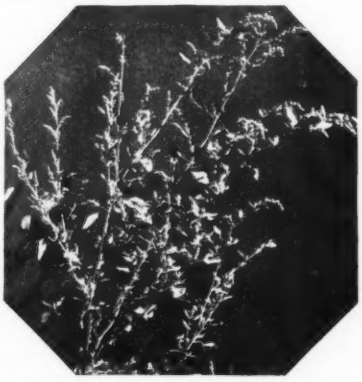
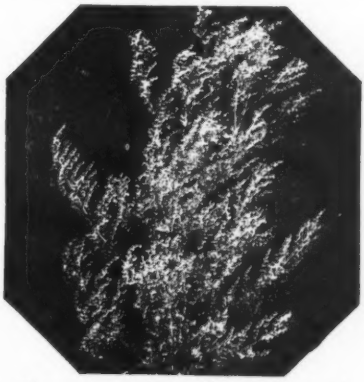
Hay Fever requirements



Artemisia annua (Annual Wormwood)



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To meet your every need in the trying hay fever season, the Mulford Biological Laboratories have produced Pollens Dried and Pollen Extracts from the hay fever plants common in your community.

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A Maltose and Dextrins

Milk Modifier

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Mellin's Food

Made from wheat flour, wheat bran, malted barley and bicarbonate of potassium — consisting essentially of maltose, dextrins, proteins and mineral salts.

Constipation in Infancy

The Fact that Mellin's Food makes the curd of milk soft and flaky when used as the modifier is a matter to have in mind when it becomes necessary to relieve constipation in the bottle-fed baby; for tough, tenacious masses of casein resulting from the coagulation of ingested milk, not properly modified, is a frequent cause of constipation in infancy.

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It is characteristic of most babies fed on milk properly modified with Mellin's Food that they are not troubled with constipation.

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Boston, Mass.

Please send "Constipation in Infancy" and "Formulas for Infant Feeding."

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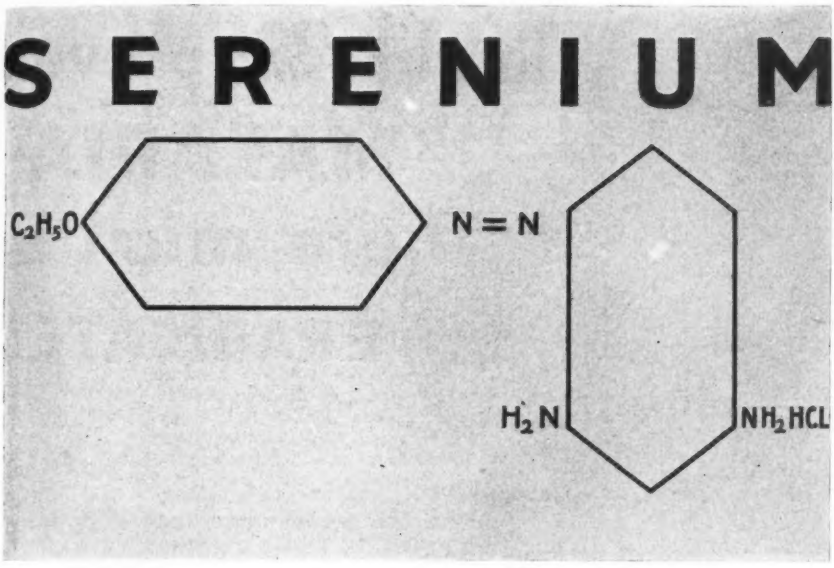

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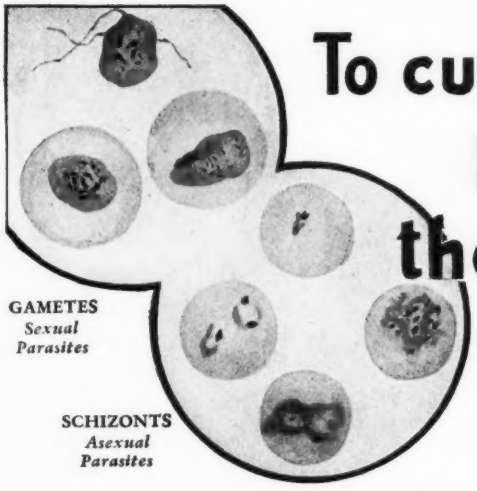

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CHINOPLASMIN DOSAGE: Adults, 6 to 8 tablets daily—Children 1 to 5 years old, 1 or 2 tablets daily—Older children, 3 or 4 tablets daily. Chinoplasmin is best given after meals. Treatment is continued for two to three weeks.

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Literature on request

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The nature of your practice will determine which of the fourteen models of Victor Mercury Vapor Quartz Lamps is best suited to your needs. If a considerable number of patients must be treated during the day, the individual treatment may be expedited by the use of the high intensity lamp, which is capable of producing a first degree erythema on an average individual in from 30 to 45 seconds, at a distance of 40 inches from burner to skin surface. Even higher intensities than this may be obtained by simply increasing the voltage to the burner. The same lamp may be adjusted so as to also obtain lower intensities when desired.

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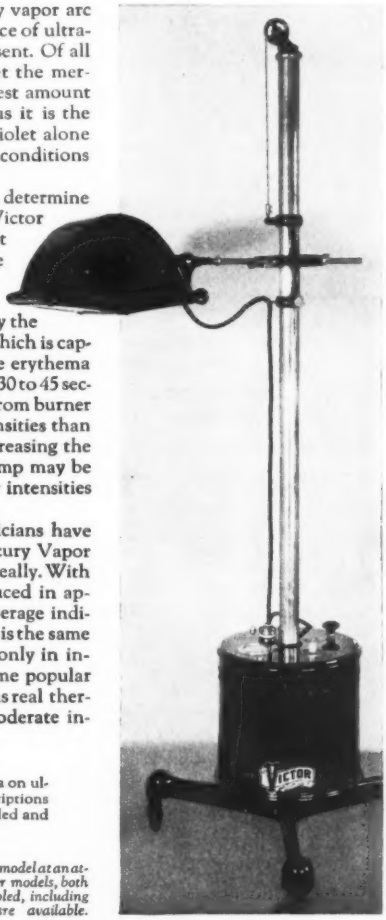

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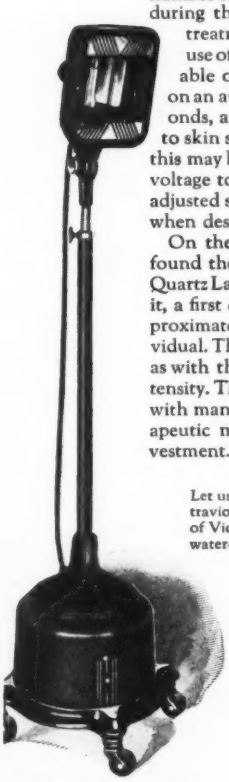

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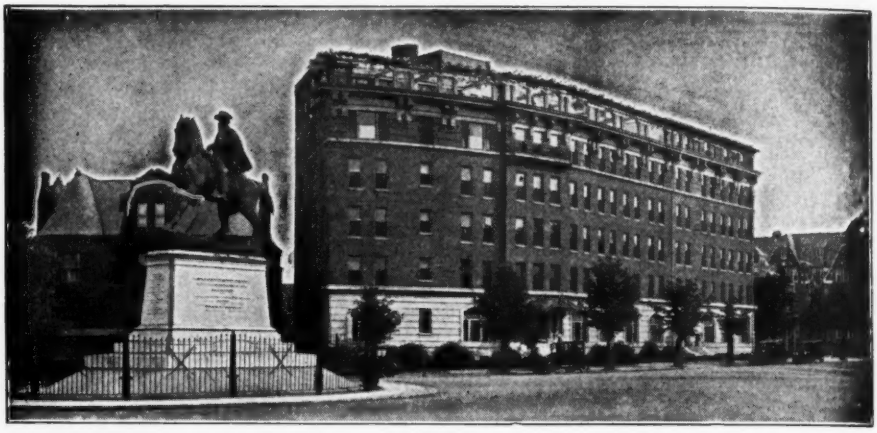

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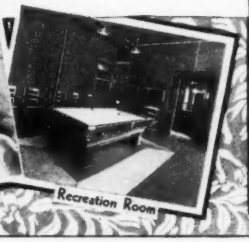
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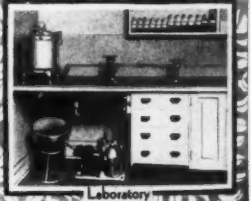
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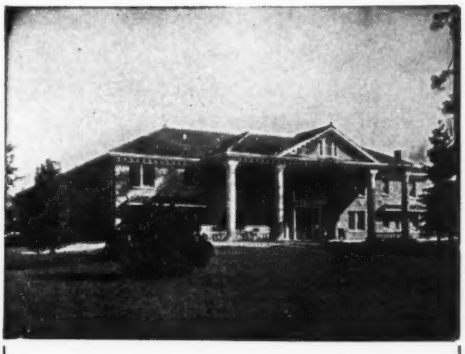

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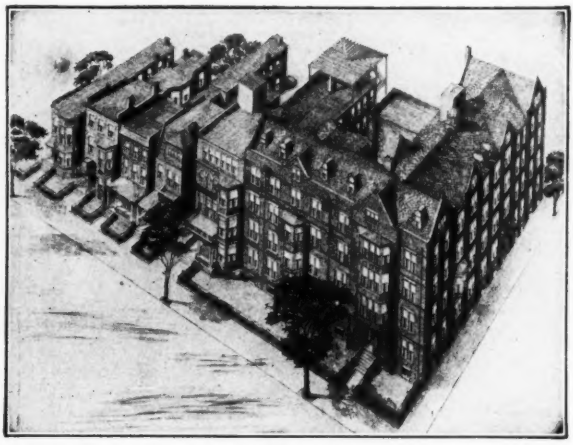

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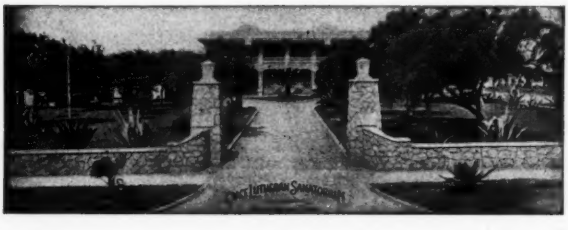

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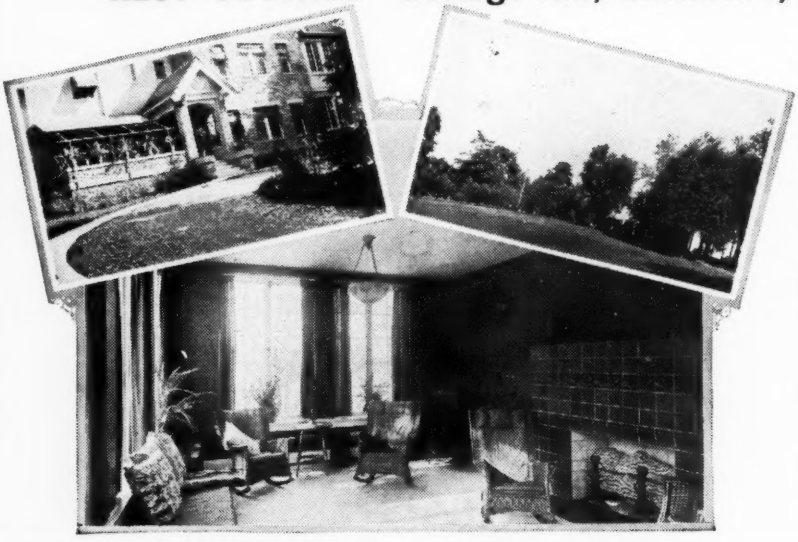

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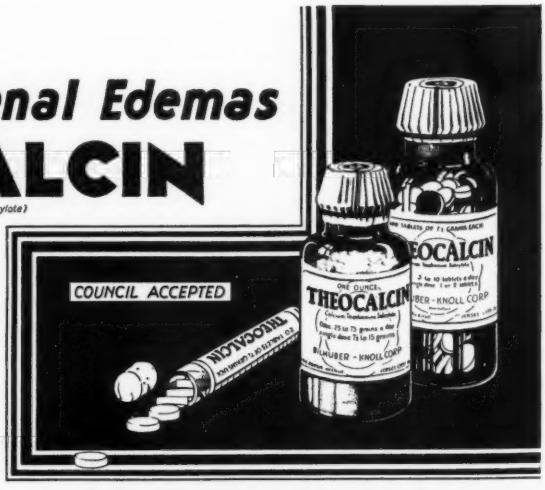

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Volume XXV MARCH 1932 Number 3

MASSIVE BONE GRAFTS: REPORT OF CASES*

By OSCAR L. MILLER, M.D.,
Charlotte, N. C.

Bone grafting is now a well established and accepted specific procedure in surgery. It is indicated mainly in the repair of defects of long bones; for stimulating osteogenesis in certain cases of delayed union; as internal splinting and support for the protection of diseased areas of bone affected by tuberculosis; and for stabilizing certain pathologically mobile joints.

Certain well-taught principles should be followed in arriving at a decision to do a bone graft. Many so-called bone graft operations have been done which might have been avoided. This is particularly true in reference to cases of moderately delayed union of bone following fracture.

A successful bone graft operation, particularly when applied to defects in long bones, depends upon the recognition of fair physiological principles, the mechanical management of the graft and the parts being repaired. The soft parts should be protected from trauma as much as possible. Attention must be given to internal and external fixation and to proper protection of the defective member for a considerable period of time.

There is still some difference of opinion as to the various types of bone best suited for particular kinds of graft, but most workers agree that the autogenous bone graft is preferable in the large majority of instances. The character of graft indicated in a given case is purely a question of judgment for the operating surgeon. All types, kinds and sizes of grafts have had their supporters. Operations have succeeded and failed for each. Whether or not a bone graft operation succeeds depends on a number of factors, and only some of these are known. Success will sometimes be attained where the odds appear greatest against it, and failure be unexplained under apparently ideal conditions.

Laboratory students are still theorizing on and debating the real physiology of bone repair and the "fate of the graft". These rather academic questions are interesting and important to the subject as a whole, but do not influence seriously the essential teaching and practice relative to the mechanical application of bone graft surgery where it is indicated. All admit that periosteum plays a useful role in bone repair. This fact should be taken advantage of in bone transplantation by preserving, as intact as possible, such periosteum as may be present in the affected area, and by bringing into the defect along with the bone graft as much periosteum as is practicable. Bone grafts, though, without any transposed periosteum have lived and contributed to regeneration of bone in the repair of a defect.

A massive bone graft is preferred for bridging a defect in a long bone, since it can be so embedded as to become an internal splint as well as direct the course of new bone growth. At the same time, experience has taught that more active osteogenesis may be expected if a large graft is reinforced along its course with just as many fine osteo-periosteal particles as can be well covered by the adjacent soft parts. All presenting dead space in the graft bed should be packed with fine bone particles. It has been proven that new bone develops along the path decreed by the bone it is to replace and by the function demanded of it. No limiting membrane is necessary to prevent new bone growing wild or in any other direction than that demanded by the body function.

Leriche, Poligard and Moore, in their treatise, "Physiology of Bone," say:

"In man, a fragment of bone transplanted into another bone always dies. At the present time this fact is a certainty. Its nutrition is no longer assured after

*Chairman's Address, Section on Bone and Joint Surgery, Southern Medical Association, Twenty-Fifth Annual Meeting, New Orleans, Louisiana, November 18-20, 1931.

its transport into the tissue of the host. In spite of its death, the transplanted fragment contracts an intimate adherence to the bed in which it is placed. This adherence is at first fibrinous, then fibrous. In this case it is currently said that the graft has taken.

"Bone tissue, having a low vitality, does not survive complete interruption of its circulation. Its nutrition is precarious and difficult. The mere fact of its having been freed from the surrounding tissues in all likelihood suffices in many cases to cause its death."

Key, a collaborator with the authors of "Physiology of Bone," believes that

"The low vitality of bone tissue renders it more liable to survive complete interruption of its circulation than would a tissue of higher vitality. Instead of the question of the death of the transplant being considered solved, as some teach, it is highly probable that transplanted bone has a better chance of living than do transplants of almost any other tissue. The truth of the matter probably lies between the two extremes; in other words, it is probable that in transplants of small bone fragments attached to the periosteum all of the transplant lives, while in transplants of large pieces of bone it is probable that cells in the interior of the transplant eventually die, while those near the surface continue to live and function. It is for this reason that transplants of living autogenous bone are more often successful than are transplants of dead bone."

Interesting as the discussions and theories may be relative to the biological behavior of bone grafts, it is still a laboratory subject, and the rank and file of us who have not the turn, nor possibly the facilities, for modern research should concern ourselves with the thus far known principles of bone graft surgery and try to apply them. Since these principles are generally well spread over the written page to a degree that any one who runs may read, it is unnecessary to attempt to detail them here.

The early work of Albee in contributing mechanical devices for obtaining massive bone grafts in the best shape and also influencing us to take considerable license with tissues in applying grafts should always be recognized and appreciated. Though some modification of Albee's teaching has taken place, it is safe to say that many failures in bone grafting have occurred because of the timidity of the surgeon in approaching his task.

While the principles of bone grafting, as now understood, are generally set forth in text-books and miscellaneous surgical literature, it must be remembered that cases where bone graft surgery is indicated by no means fall into such simple classifications that perfect end results may be expected to follow literal academic instructions. That is, the successful conclusions of a case where a radical bone graft operation is required often calls for all the surgical acumen one can bring to bear, and the surgeon may occasionally feel that he should have consulted the oracles in addition.

The possible value, then, one may hope for in reporting a series of cases of massive bone grafting at this period of progress in the subject is to bring out interchange of experiences in the approach to individual problems as they present themselves in the run of a surgical practice. As stated before, one may experience easy success in what appears to be a difficult bone graft and unexplainably fail where it appeared he should have succeeded easily. In the treatise, "Physiology of Bone," formerly referred to, it is well said

"The rehabilitation, rarefaction, and reconstruction of a transplant are delicate operations that we do not know how to control and which always allow some incertitude to arise as to the therapeutic result in view of the reason for undertaking the transplantation."

Because of its character and accessibility, the tibia has become the most popular bone from which to secure autogenous bone grafts of every kind. Other bones, as the fibula, the ribs, the ilium, are used in special instances, but these bones do not supply massive grafts comparable to those available in the tibia.

The operation of massive bone transplant disturbs, to some degree, the normal body physiology and, as in other major operations of election, one wishes to have a patient in the best possible physical condition. Many, really most of these operations, are required in subjects whose defects have arisen from osteomyelitis. There is always the possibility of recurring infection in such cases. Having given the field of operation a reasonable period of healing and attenuation of organisms following osteomyelitis, one may still succeed with a bone graft in the presence of recurring infection. However, such an experience is far from ideal and may be an harrowing one for both patient and surgeon. Where the epiphyses have been spared by an osteomyelitis, or other affections, and bone graft surgery is required to bridge a resulting defect, very normal appearing and functioning limbs may be recovered. As compared to the period when amputation for such conditions was a common practice, this advancement is encouraging.

Once a bone transplant has taken, its progress

should be checked at intervals by x-ray examinations and motion of the parts instituted reasonably early as a therapeutic measure. In a lower extremity, weight bearing in a protecting leg cast, and later a brace, stimulates growth and hastens the termination of treatment. These measures have been commonly practised in this series of cases.

CASE REPORTS

Case 1.—F. B., aged 5, presented an ununited fracture of the left tibia of three years' duration. At age two, while under treatment for rachitic bow-legs by use of pressure in plaster casts, she sustained a fracture of the left tibia at the junction of the middle and lower thirds. An open reduction of the fracture had been undertaken, but non-union ensued. Five months later the fracture site was again opened to effect union and this attempt was unsuccessful.

The patient was first seen at the clinic of the North Carolina Orthopedic Hospital, October 1924, with a defect in the left tibia, and unable to bear weight on the affected limb. Because of the child's rather poor physical condition and the comparatively recent disturbance of the tissues at the fracture site by operations, further surgery was not advised at this time. A light leg brace was applied and the child encouraged to walk on the limb. This she did for almost a year and a half. During the interval, while reporting to the outpatient clinic and walking in the brace, she recovered from evidences of rickets, but developed considerable atrophy of the defective tibia and a deforming overgrowth of the fibula.

In April 1926, this patient was admitted to the hospital for a bone graft operation. It was necessary to do an osteotomy of the fibula to bring the ends of the tibia into proper alignment. A graft bed was prepared in the defective tibia by splitting the two eburnated fragments from their pointed ends well toward each epiphysis. Care was exercised to preserve all possible surrounding tissue attachments to the periosteum of the fragments. With an Albee saw, a substantial wedge-shaped graft was taken from the sound tibia and dovetailed snugly into the prepared bed. Some small shaving grafts were packed along the point of greatest defect in the tibia. A protecting leg cast was applied.

Six weeks after operation, the child was allowed up on crutches. X-ray check on the progress of the graft at this time demonstrated union beginning from both ends of the tibial fragments. Weight bearing was allowed in a light leg brace ten weeks after operation and future convalescence was uneventful. Five years after bone graft operation the patient has a normal appearing and functioning limb with one-half inch shortening.

Case 2.—S. E. was a boy of 5. At the age of 3 he had a radical operation for acute osteomyelitis of the left tibia with so much loss of bone substance that non-union ensued. He was presented for a bone graft operation two years later. All the osteomyelitis was well healed.

X-ray examination showed long atrophic eburnated fragments and a flail tibia in the mid-third. There was a fair rate of growth in the tibia as compared with the fibula, in spite of the non-union. The left leg was two inches shorter than the right.

The patient was admitted to the hospital and prepared for operation. The fibula was osteotomized to allow improvement of alignment of the tibial fragments. With an Albee saw, a bone graft six and one-half inches long was removed from the sound tibia and securely wedged into the prepared bed and split ends of the tibial fragments. Some fine osteo-periosteal particles were placed in any dead space along the graft bed. A snug-fitting leg cast was applied as a convalescent dressing.

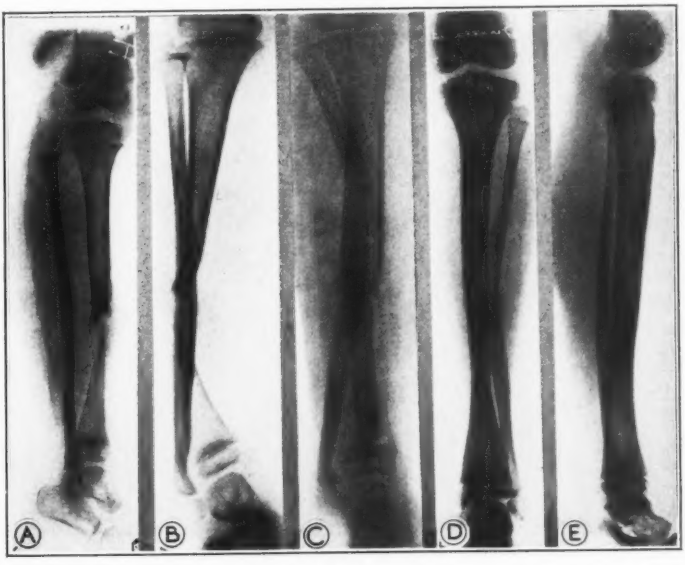

Fig. 1

Case 1.—A, Defect in tibia following osteoclasis and operations for non-union in rachitic bone. B, Deformity at ankle from overgrowth of fibula. C, Six weeks after bone graft operation. D, Appearance of tibia eight months after operation. E, Five years after bone graft.

Fig. 2

Case 2.—A, Non-union, left tibia, following osteomyelitis: extensive eburnation and atrophy of fragments. B, Ten weeks after bone graft operation. C, Two years after operation.

The wound healed promptly. X-ray examination at ten weeks showed the graft taking satisfactorily and a walking cast was applied. Unprotected weight bearing was allowed seven months after operation. The patient was last seen two years after operation. Function of the leg was good with three-fourths inch shortening.

Case 3.—L. H., a girl of 12, in 1923 had had an operation for osteomyelitis. The lower third of the shafts of both tibias were resected in the acute stage, resulting in extensive loss of bone substance and non-union. Following the operation for osteomyelitis, considerable deformity occurred at the ankles due to continued growth of the fibulae and the defect in the tibiae.

In 1926 this patient, unable to bear weight on the limbs, was admitted to the hospital and prepared for a bone graft operation. The first step was osteotomy of each fibula and forcible correction of the mal-alignment of the tibial fragments. After an interval of a few weeks, the first bone graft operation was done on the left tibia, it having the longest defect. With an Albee saw, a substantial piece of bone was removed from the right proximal fragment. The lower tibial fragment was split as near to the ankle joint as one dared go; the upper fragment was split until good bone marrow was seen and the graft was tightly wedged into place. There was a moderate amount of tissue disturbance following this operation, suggesting reactivation of the original infection, but the wound soon healed and the graft took promptly.

Two months after the first bone graft operation, the defect in the right tibia was corrected. The bone used here was taken from the long proximal tibial fragment above, which furnished the first graft. Convalescence from the latter operation was uneventful. X-ray examination made in a few weeks demonstrated successful take of both grafts.

Protected weight bearing was allowed at the end of six months. The patient has been followed for five years. X-ray films reproduced in connection with this case report show the general progress of the bone grafts. The child has excellent function in both limbs.

Case 4.—C. J., a boy of 12, was presented in 1928 for bone graft operation. Sixteen months prior to this

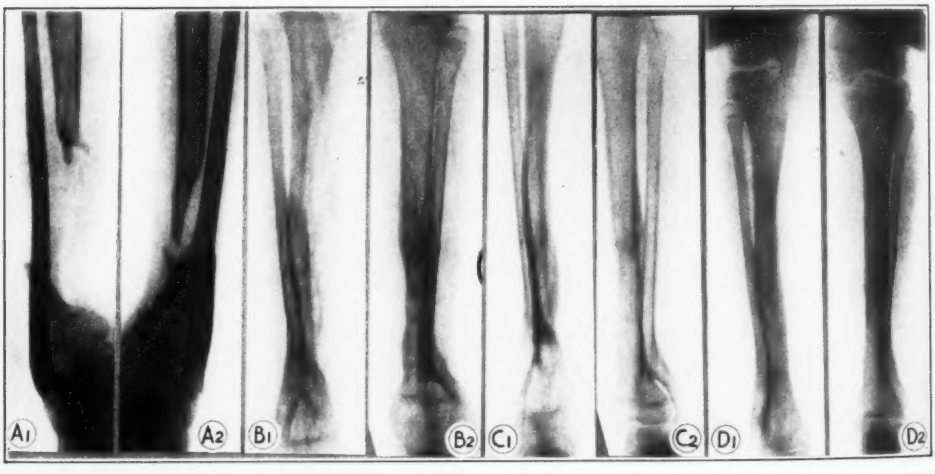

Fig. 3

Case 3.—A1 and A2, Defect in both tibiae following resection of shafts for acute osteomyelitis; growth of the fibulae contributed to deformity at the ankles. B1 and B2, Five months after operation on left tibia; four months after operation on right tibia. C1 and C2, One year after operations. D1 and D2, Appearance of tibiae three years after operations.

Fig. 4

Case 4.—A, Defect in upper half right tibia following diaphysectomy for acute osteomyelitis. B, Two months after bone graft operation. C, Appearance of graft five months after operation.

date, in a neighboring hospital, diaphysectomy of the upper half of the right tibia was done in the treatment of acute osteomyelitis.

The fibula was first osteotomized in its upper third to permit easy alignment of the tibial fragments. A graft seven inches in length and three-fourths inch wide, slightly pointed at each end, was removed from the sound left tibia by means of an Albee saw. This mass of bone was placed along the defect in the right tibia and securely driven into the split ends of the fragments. Some small osteo-periosteal fragments were packed around the graft and the whole securely covered by surrounding soft parts. The limb was dressed in protecting plaster cast.

There was some tissue reaction and low grade infection following operation. This cleared up reasonably soon and further convalescence was uneventful. Regeneration of bone across the defect was rather rapid and substantial. Protected weight bearing in a cast was allowed three months after operation and weight bearing in a brace two months later.

This patient has been followed for two years and now has a useful weight bearing leg. Under the stress of function much of the shortening is being corrected.

Case 5.—T. O., a boy, aged 13, was admitted to the hospital for correction of a defect in the lower half of the left tibia. Two and one-half years prior to this time, in a neighboring hospital, the child had resection of a large portion of the shaft in the treatment of acute osteomyelitis. Some overgrowth of the fibula gradually threw the foot and lower fragment of the tibia into varus deformity. There was a large gutter scar over the old diseased area in the tibia. No drainage had occurred here for over a year.

The first step in the management of the case was to resect the old scar, do an osteotomy of the fibula and bring the lower tibial fragment into alignment with the upper one. Tissues hea'ed promptly following this operation. Two months later, at a second operation, a fragment of bone eight inches in length was removed from the sound tibia and securely wedged into the split ends of the tibial fragments to bridge the defect. The leg was dressed in a strong plaster cast.

Four or five days after operation the patient developed high fever, chills and every evidence of infection. A window was cut in the plaster cast to expose the wound. A postoperative osteomyelitis was in evidence and copious drainage of pus occurred. This drainage continued for months, but fixation of the leg in the cast was constantly maintained. The large bone graft became a sequestrum and was removed from the wound four months after operation. The drainage gradually cleared up and a very well proportioned tibial shaft developed across the defect in spite of extensive wound infection.

Protected weight bearing was allowed at seven months and unprotected weight bearing at the end of a year. Some delayed union occurred in the lower aspect of the hiatus, but under the stress of weight bearing,

Fig. 5

Case 5.—A, Defect in lower half left tibia following resection of shaft in the treatment of acute osteomyelitis. B, Appearance of graft six weeks after operation in the presence of active infection. C, Four months after operation; the graft is a sequestrum; much bone has formed beneath it. D, Appearance of tibia two and one-half years after bone graft operation. E, Four years after operation; good weight bearing tibia.

Fig. 6

Case 6.—A, Pathological fracture and non-union left femur following osteomyelitis; four and one-half inches shortening. B, Twenty-one months after operation; one and three-fourths inches shortening.

bone eventually bridged this defect. The lower tibial epiphysis appeared to have been destroyed by the original infection, and there were two inches of shortening in the leg when the period of treatment terminated.

This patient was seen four years after operation at the age of eighteen. The tibia had united. He had slightly over one inch shortening of the leg. This has been compensated for by a raised shoe. He had twenty-five degrees of ankle motion and his gait was excellent.

Case 6.—A. C., a girl of 6, was admitted to the hospital because of a defect in the left lower extremity following osteomyelitis of the femur, which had occurred some years before, with four and one-quarter inches shortening of the left leg. In spite of the defect in the femur and the shortening, the child bore weight on the affected limb, but she had an ugly limp. All draining sinuses were healed and the child appeared physically fit for operation on admission.

X-ray examination showed a pathological fracture and non-union in the middle third of the left femur; also, much atrophy and overlapping of the pointed fragment ends.

Before operation was undertaken, a Hoke traction apparatus was applied and some two inches in length gained for the defective limb. The operation consisted of freshening the eburnated ends of the femoral fragments for several inches above and below the defect and fastening these fragments to one another with a kangaroo tendon suture to prevent recurrence of overriding. A large mass of heavy osteo-periosteal grafts were then fitted snugly between and around the fragments for a distance of about three inches above and below the fracture site. A snug-fitting plaster spica was applied as external fixation.

Union was rather slow in this case, but did occur, and protected weight bearing was allowed six months after the bone graft. The patient was dismissed from the hospital nine months after operation with a strong bony bridge developing over the defect. The gait was improved and there was slightly over two inches shortening. Much more of the length should be recovered in a few years under the stimulation of function.

Case 7.—L. C., a woman, aged 30, sustained a fracture at the junction of the middle and lower thirds of the left humerus in an automobile accident in July 1928. X-ray examination made at that time showed very good approximation of comminuted fragments and it appeared that union should have occurred under ordinary conditions. The arm was placed in extension for several weeks in a Jones arm splint. At the end of two months there was no union nor evidence of callus deposit.

It should be noted that just prior to the accident this patient had been on a severe reducing diet and had reduced her weight rather rapidly by twenty five pounds. This may have had something to do with the failures of the humerus to throw down callus.

Open operation and bone graft were decided upon and

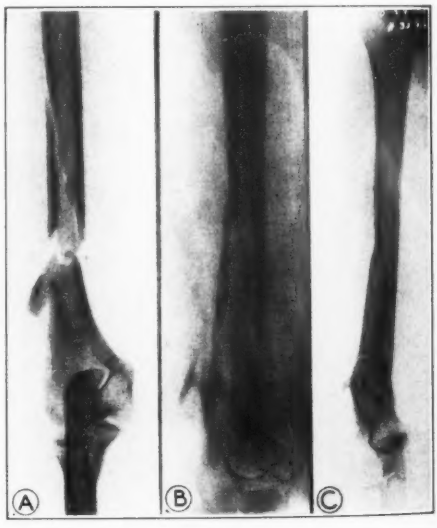

Fig. 7

Case 7.—A, Fracture, lower third, left humerus with non-union. B, Bone graft wedged into split ends of humeral fragments. C, Appearance of humerus three years after operation.

this procedure was carried out September 22, 1928. The fractured fragments in the humerus were exposed and split with a chisel to receive the graft. A substantial piece of bone, five inches in length, moderately pointed at each end, was taken from the left tibia by means of an Albee saw and securely wedged into the prepared bed at the site of fracture. Some fine osteo-periosteal particles were packed in around the large graft. A plaster of Paris shoulder spica was applied to this patient before operation and served as a convalescent dressing during the healing period.

X-ray examination made shortly after operation showed the bone graft in good position and alignment of bone fragments satisfactory. Within two months, sufficient union had taken place to permit guarded exercises directed toward recovering the function of the elbow joint. The shoulder spica was removed shortly thereafter. Further convalescence was uneventful.

At this time, three years after operation, the patient, an operating room nurse, has a strong and normally functioning arm.

Case 8.—R. M. was first seen at the Clinic of the North Carolina Orthopedic Hospital, at age nineteen months, with a congenital anterior bow in the right leg. He was beginning to walk, bearing some weight on this extremity in spite of the deformity.

At the age of two and one-half years he sustained a fracture at the junction of the middle and lower thirds of the right tibia. Leg cast was applied to hold the fragments in the best possible alignment. The child was allowed to walk in a cast in the hope that this would stimulate callus deposit and possible union of the fracture. The cast was removed and the leg inspected at the end of two months. There was no evidence of union.

A bone graft operation was done May 30, 1930. A full thickness bone graft about six inches in length was removed from the left tibia and securely wedged into the prepared bed and split ends of the fragments of the right tibia. It was noted at the time of operation that the character of the tissue at the site of the break suggested the type of tissue usually seen in congenital fractures. A snug-fitting leg cast was applied as external fixation.

X-ray examination made during the next few months showed the graft uniting to the large proximal fragment, but absorption beginning around the graft in the lower fragment and fracture eventually occurred at the level of the original break. Fragments started angulating again in the direction of the old deformity. In spite of the fact that this graft failed to take, it appeared eventually that both tibial fragments showed better type of bone substance.

On February 9, 1931, a second bone graft was done. This differed very little from the former operation except probably a longer incision was made and more freedom exercised in splitting the tibial fragments. A substantial bone graft was cut from the sound tibia by means of an Albee saw and wedged very snugly into the bed prepared along the posterior aspect of the right tibia.

This latter graft was made to conform very much to the line of a normal tibia and contracted firmly in the upper fragment, and in the lower fragment it passed entirely over the epiphyseal line. In preparing the bed for receiving the transplant there was much fragmentation of the old tibia. A number of osteo-periosteal

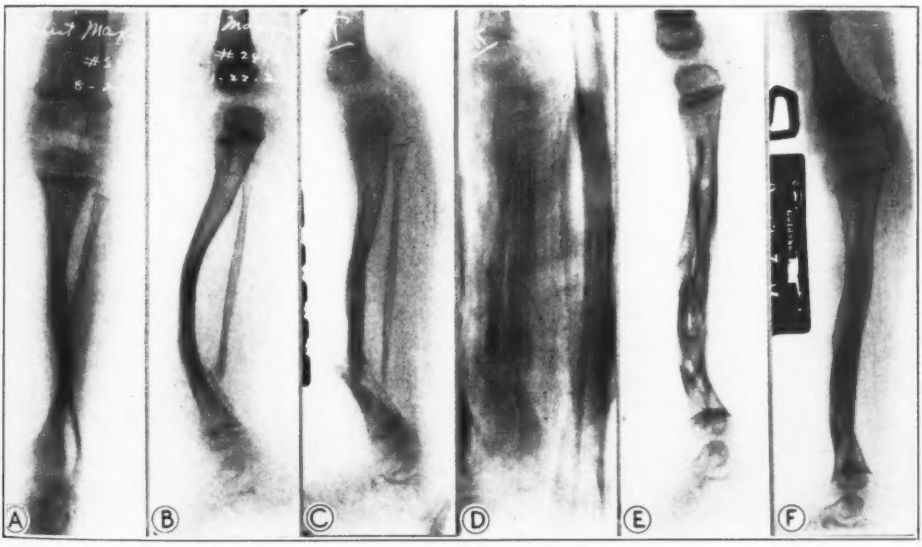

Fig. 8

Case 8.—A, Congenital anterior bowing of right tibia. B, Fracture in lower third of defective tibia. C, Appearance of tibia after failure of first bone graft operation. D, After second operation; large bone graft placed somewhat posteriorly, forcing tibial fragments into alignment. E, Three months after successful bone graft. F, Appearance of tibia eight months after bone graft.

particles were placed along the line of expected bone development. A light snug-fitting plaster cast was applied for protection during convalescence. The wound healed promptly.

X-ray examination made ten weeks after operation showed the bone graft extending practically the entire length of the right tibia, the lower end apparently invading the epiphysis and union taking place. At this time considerable proliferation of new bone could be seen all along the course of the new graft.

Three months after operation the child was allowed to exercise the leg in bed without weight bearing. At fourteen weeks protected weight bearing was allowed. X-ray examination, five months after operation, demonstrated strong union and regeneration of practically a new tibia along the graft and much improved alignment of the shaft of the tibia. The fibula remained ununited in its lower third.

X-ray examination eight months after operation showed a very normal looking tibia developing and no evidence of tendency to recurrence of fracture. Full weight was then allowed in a leg brace.

Fig. 9

Case 9.—A, Defect in right tibia following diaphysectomy for acute osteomyelitis. B, Four months after implantation of bone graft. C, Nine months after operation. D, Appearance of the tibia three years after operation.

Case 9.—P. F., a girl, aged 9, was admitted for repair of a defect two years after diaphysectomy of the right tibia in the treatment of acute osteomyelitis. There had been no active infection in the leg for over six months.

Preliminary to bone graft operation, a long gutter scar was resected from the lame leg and an osteotomy of the fibula was done to improve alignment of the tibial fragments. Recovery from this procedure was uneventful and a few weeks later the patient was prepared for a bone graft. At operation, a bed was prepared along the defect from beyond the epiphysis in each fragment, as shown in the x-ray films illustrating the case. A long substantial graft, including some of the substance of the upper and lower epiphysis, was then taken from the sound tibia. This graft was securely wedged into the remaining fragments of the old resected tibial shaft and extended upward and downward across the epiphyseal lines of each. Healing was attended by low grade superficial infection and extrusion of some small sequestra, but the large graft survived very satisfactorily.

Progress of growth in the regenerating shaft of the tibia was checked by x-ray pictures at intervals. At four months, protected weight bearing was allowed in a snug-fitting leg cast. Protection was continued for one year, at the end of which time a very substantial tibia had formed along the entire diaphyseal defect. This patient has been followed for three and one-half years since operation. She has a good weight bearing limb with one-inch shortening.

PLASTER CAST, BONE PIN METHOD IN FRACTURES OF THE LOWER LEG*

By J. WARREN WHITE, M.D.,
Greenville, S. C.

The purpose of this paper is to describe a way of caring for difficult fractures primarily of the lower leg, which would ordinarily require prolonged traction or some method of internal fixation. The method cuts short hospitalization and in a definite number of cases obviates an open operation.

Since I elected the above mentioned subject, Pitkin and Blackfield,[1] of San Francisco, have written an article describing the essential principles employed, but differing considerably in the method of application. Bohler,[2] in his os calcis paper which appeared in January, used the same idea, but applied it only to fractures of the os calcis. About a year ago Speed,[3] of Chicago, published an article on plaster embedded

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*From the Shriners' Hospital for Crippled Children, Greenville, South Carolina.

skeletal traction, but only a single pin was employed.

In a recent publication Bosworth,[4] of New York, came out with an excellent article on "Skeletal Distraction," which is a most apt term when one recalls that the first meaning of the word *distract* given in Webster's Unabridged, is "tearing asunder." Bosworth does not employ plaster, but does use a splint similar to Abbott's[6] tibial lengthening apparatus.

In my paper[5] on "Tibial Lengthening," published in January, 1930, I mentioned the applicability of the technic therein described to the treatment of certain fractures, although at that time I had not made use of it in that way. There is really little essential difference between the method described in this tibial lengthening article and the technics described by Bosworth[4] in April, 1931, and Pitkin and Blackfield[1] in July, 1931. All three methods are founded on the technic made practicable by Abbott.[6]

It is with the method of application that this paper will deal. It is suited *par excellence,* as stated, to fractures of the lower leg, including the os calcis, but I see no reason why it cannot be applied to the forearm as well. Here again the principle of wedging may be employed which I mentioned in my os calcis article,[7] if the position as seen after the application of the cast does not seem to be satisfactory.

Those of us who have used the plaster pin method have all been dissatisfied with the difficulty encountered in removing the pin. Some undoubtedly are still using the so-called Steinman pin that unscrews in the middle. Many also must have experienced so much trouble with its breaking that it has been discarded. I had three which broke within a short space of time, and I have never used another and never intend to.

The employment of wire instead of a pin is gradually becoming popular. The removal of the wire is a relatively simple matter. It is clipped with a sterile pair of wire cutters just below its normal exit from the skin on one side and the wire is pulled through on the other side.

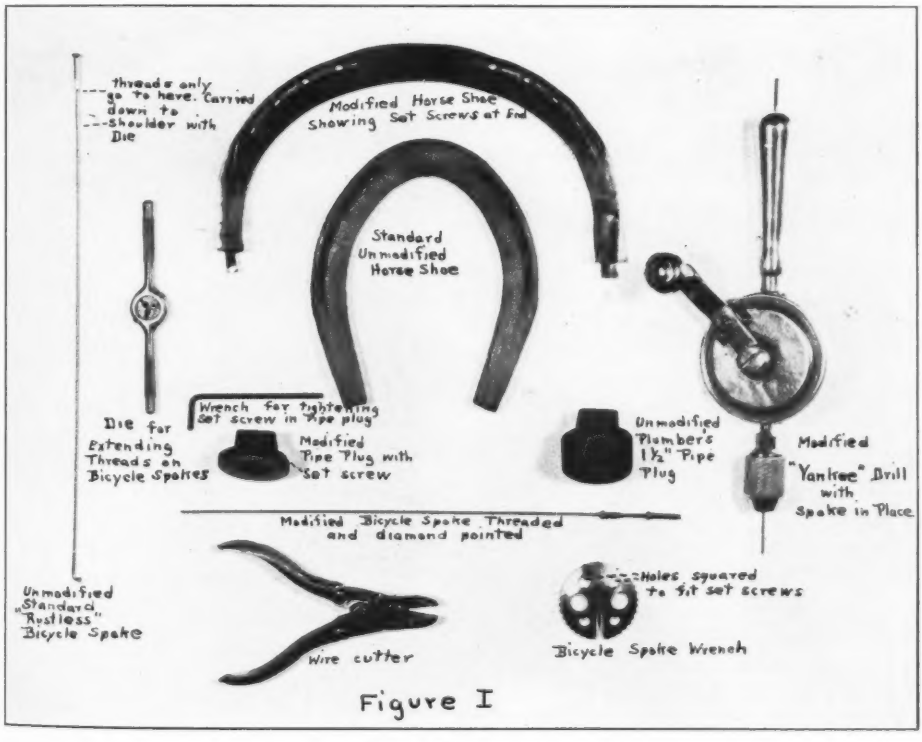
threads only go to here. Carried down to Shoulder with Die
Modified Horse Shoe Showing Set Screws at End
Standard Unmodified Horse Shoe
Wrench for tightening Set screw in Pipe plug
Die for Extending Threads on Bicycle Spokes
Modified Pipe Plug with Set screw
Unmodified Plumbers 1½" Pipe Plug
Modified "Yankee" Drill with Spoke in Place
Modified Bicycle Spoke Threaded and diamond pointed
Unmodified "Standard" "Restless" Bicycle Spoke
Wire cutter
Holes squared to fit set screws
Bicycle Spoke Wrench
Figure I

Fig. 1

The usual procedure is to paint the area, including, of course, the pin, generously with tincture of iodine where it emerges, and then, pressing down the skin as deeply as possible with the sterile cutters, to cut the wire. This technic has never, at least with me, been followed by any trouble. Pulling the pin through, however, often is a rather painful procedure to the patient; and however thoroughly I sterilize the area, I never feel comfortable about it and am always relieved when the wound heals.

For this reason I have for some time been trying to develop a scheme whereby I could substitute wire for the pin in the plaster pin method, and believe I now have a workable technic.

By experimentation I have found that a well applied plaster cast resists lateral compression enough to permit its being used as the rigid bow is used in the Kirschner wire apparatus. As lateral compression cannot be applied until the cast has thoroughly dried, a plan had to be devised that would hold the wire tight enough to withstand the force employed until the plaster was no longer green. For this I use the Kirschner wire principle with the modifications described below until the cast becomes sufficiently rigid. This usually takes about three days.

While the financial outlay in providing ourselves with the standard Kirschner wire equipment is not prohibitive, there must have been others besides myself who thought that it was rather expensive, and with the method described in this paper one has to be equipped with several more extra large "horseshoes."

Dr. Edward King, of Asheville, suggested using ordinary horseshoes. This idea appealed to me and I have been using them ever since. With the necessary spreading and squaring of the ends and nickle plating, the outlay for each shoe is a little over a dollar. It is a simple matter to drill the few necessary holes for the wire and set screws. I have done this myself.

A recent article by Mathews[8] describes a method of modifying a hand drill to replace the cumbersome and expensive extension drill supplied with the Kirschner apparatus. This was a very clever idea and I have been using, with a great deal of satisfaction, a "Yankee" hand drill modified according to his suggestions.

Another problem I encountered in employing the horseshoe was in tightening the wire. In discarding the horseshoe and drill of the Kirschner apparatus I also wanted to avoid if possible getting a wire tightener. A clerk in a hardware store whom I consulted suggested a bicycle spoke, and with the threaded end this proved just the thing. I sharpened the hub end of the spoke and continued the thread at the nipple end to give a greater excursion of the tightening nut or nipple, as it is called in bicycle parlance.

The technic of application is as follows:

The field and instruments have been prepared as for a bone operation, and the former painted with iodine in the usual way. The spokes are drilled through the bone at the places of election above and below the fracture and left *in situ*. Protective dressings are then slipped over their ends, which are then made to pierce in the center, sterile two-inch circular washers, cut from an inner tube. The washers, in addition to holding the dressings close, prevent contamination down the spoke from the outside.

Next are slipped on the spokes two cast iron arrangements cut from plumbers' 1¼-inch pipe

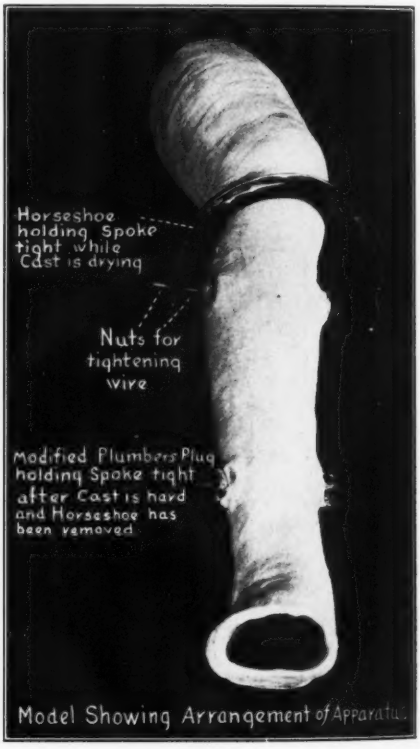
Horseshoe holding Spoke tight while Cast is drying
Nuts for tightening wire
Modified Plumbers Plug holding Spoke tight after Cast is hard and Horseshoe has been removed
Model Showing Arrangement of Apparatus

Fig. 2

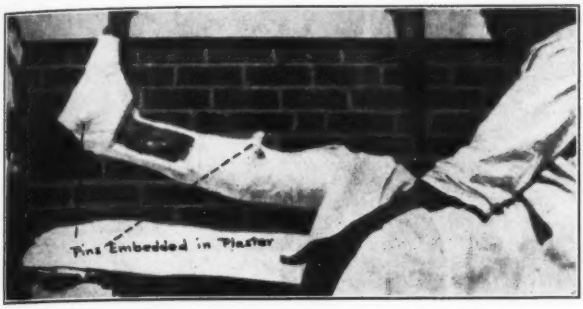

Fig. 3

Photograph of shotgun wound case with extensive loss of osseous tissue being held with pins and fenestrated cast. X-ray shown in Fig. 4.

plugs, especially prepared (*see Fig. 1*). These washers are to fit against the outside of the cast. One is fitted with a set screw to hold the wire as it passes through it and the shoulder of the spoke nut or nipple is screwed against the other at the other end.

The horseshoe is now adjusted to hold the spoke tight, using a set screw at one end and the spoke nut at the other. The two nuts at the screw end of the spoke show why all the threads possible are necessary. This is tightened, using the usual spoke wrench, to the desired amount. With the two washers on the spoke inside the ends of the horseshoe, not too close together, and employing the desired amount of traction on the distal shoe by means of a hook and chain at the tips, a plaster cast is applied, making it possibly a little thicker than usual where the wires pass through the leg. I carry the plaster well up the thigh with the knee slightly bent.

Traction on the distal shoe is maintained until the cast has set well. The inside iron washers are fitted close to the cast while it is hardening, care being taken to allow a sufficient number of threads on the threaded end of the spoke to permit extra tightening of the washers after the plaster has dried.

As stated before, the horseshoes holding the wires tight are allowed to remain on for three or four days and then just before they are removed the nut against the inside washer is tightened enough to keep the wire as tight as it was kept by the shoes. They are then removed and the excess wire clipped off.

The length of time that the wires are left in place, of course, depends on the particular case and need not be discussed here. In removing them, all that is necessary is a hacksaw to cut out a generous section of the cast on one side, holding a washer to which a wire is attached. Then, after loosening the nut or set screw, as the case may be, the section including the washer is taken out, disturbing the wire as little as possible. The underlying padding and dressing are carefully removed and the wire is clipped, as described above. Another section is cut on with the hacksaw on the other side of the cast, including the other washer. This is then taken out, pulling with it the wire which has been cut on the other side. The dressing need not be disturbed on this side until the cast is changed.

In using this method, which I admit is still somewhat in the experimental stage, one must be absolutely sure of his plaster. A soft, nonresistant plaster will not withstand the tightening necessary to prevent the wire from sagging unduly when the muscle spasm tends to pull the bone ends together. A bicycle spoke which goes under the trade name of "rustless" is satisfactory.

Fig. 4

See *Fig. 2* for a photograph of a model of the apparatus with one shoe still in place.

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DISCUSSION (*Abstract*)

Dr. W. B. Owen, Louisville, Ky.—It is necessary to reduce the cost of apparatus in orthopedic surgery, and this is an unique, simple and cheap arrangement. It is, of course, to be employed in selected cases in which skeletal traction is necessary over a prolonged period of time.

The principles employed are very similar to those of Dr. Lorenz Bohler, who was in this country recently.

I am opposed to many open operations in fractures because of the fact that probably 90 per cent of our non-union cases result from or occur after an early primary open operation. This method simplifies the treatment of this particular type of case and makes it safer for the patient.

Dr. W. K. West, Oklahoma City, Okla.—Skeletal traction, when used as fixed traction, as described in this paper, is somewhat similar to the Kirschner wire method that has been used for several years except that the apparatus is not expensive. We have been using the Kirschner wire method in treatment of fractures of the femur and of the tibia since I visited the Clinic of Dr. F. J. Gaenslen, of Milwaukee, a few years ago. While the Kirschner wires, bows, wrenches and drills are expensive, they are well worth the price to any one doing a large fracture practice.

I think the demonstration has been exceedingly interesting. In these particular cases the bicycle spoke arrangement would allow the patient to be ambulatory much sooner.

There is just one thing more I wish to leave with you. In my travels about the country within the last two years, in discussing the use of the wire with the different men, I find that many of them hesitate to use this type of traction because of the danger of the wire's cutting through the bone. We have not seen a single case in which that has happened. The method has also been criticized on the ground that the small wire would cut into the periosteum and be painful, but this is not true. On the other hand, the wire is so secure that the patient moves up and down in bed without the pain that we often notice when using the ice tong method, which is especially noticeable in women of a nervous type.

From my experience with this type of traction I believe that the patient with a fractured tibia, treated by Dr. White's method, would be perfectly comfortable.

Dr. E. D. Fenner, New Orleans, La.—In the country at large there seems to be a great difference of opinion as to the use of open reduction or the closed methods. Those who are working here seem to be very strongly in favor of closed methods in recent fractures. In other parts of the country open reductions are more popular.

No matter how skillful or careful the surgeon may be, when he does an open reduction on a fracture, he certainly increases the danger to the patient, and I believe that this method of pin-plaster of Paris reduction will render possible an entirely satisfactory reposition of 90 per cent of the fractures that we are called upon to treat in regions where the application of the pin is possible.

Dr. H. Earle Conwell, Birmingham, Ala.—There is one warning which I wish to make regarding the use of skeletal traction in the treatment of fractures. Some surgeons use skeletal traction with the intention of leaving the skeletal traction in the bone until a firm union has taken place at the site of the fracture. From this prolonged use I believe that more complications of osteomyelitis or local inflammatory conditions result.

I use skeletal traction only until the soft structures have improved to such an extent that a simple fracture has been more or less produced, and until there is sufficient callus formation at the site of the fracture to prevent mal-position. Following this, a simple fixation in the form of a plaster cast or its equivalent is applied.

The question of whether we shall do an open or closed reduction, whether the latter shall be by skeletal traction or otherwise, depends upon the following considerations. It is not a question of whether the surgeon should always do an open or closed reduction in a fracture, nor is it a question for argument whether one will get an infection in an open reduction; but the surgeon should try the conservative method of reduction first in the majority of fractures, and if he is not successful, he should be capable of performing further operative procedures. Treatment of fractures not only includes the treatment of bone, but muscles and nerves and blood supply and other soft structures.

One's ability as an orthopedic or general surgeon in treating fractures is not determined by the number of open reductions which he does with or without infection, but by the functional results obtained, whether by open or closed methods.

Dr. White is to be congratulated on his apparatus and we should profit by its use.

Dr. White (closing).—Time and again I have sent my patients home with the pins in. At the present time I have two at home, out of the hospital, with the pins in. I only keep them in the hospital until they are comfortable. If there is some discomfort that shows that something is wrong, I straighten that out before they are discharged. In one case I left the pins in for ten weeks and we found no reaction on removing them at all. In fact, in a tibial lengthening case in one instance four pins were in one leg for twelve weeks without complications.

In the x-ray of that second case, which I showed, a good many of us would not have been satisfied with the position obtained. The patient was a colored woman, 65 years old, to whom an anesthetic could not be given and who, I think, will get, with that position, a very satisfactory leg. She is the patient who at the present time has the pins in and is living at home. Of course, she is seen every week at the clinic.

ECZEMA IN INFANCY AND CHILDHOOD: ITS RELATION TO ALLERGY*

By LEE BIVINGS, M.D.,
Atlanta, Ga.

The importance of allergy as an etiological factor in the dermatoses of infancy and early childhood is being accepted both by dermatologists and pediatricians. Reports of success in relieving long-standing eczemas by treatments taking this factor into account appear more and more frequently in the medical literature of all nations.

From a practical standpoint, the skin of infants and small children reacts more readily and with a larger number of diagnostically positive tests than the skin of older children or adults. Another factor of importance is that most eczemas of infancy are apparently due to foods, which makes the search for the causative factor much simpler.

Recent research has given us a scientific basis for the theory of allergy as applied to eczema. Kolmer[1] believes that capillary permeability is the hereditary factor in allergy. O'Keefe[2] thinks that capillary permeability is the result of protein sensitization. Walzer[3] has shown that the absorption of the unaltered protein molecule is a natural phenomenon and contact with sensitized groups of cells brings about reactions. Ratner, Jackson and Gruehl[4] demonstrated both active and passive sensitization of the fetus *in utero*, and Ratner[5] has gone further to show a causal relationship between the antepartum diet of the mother and the reactions of her child. Stuart and Farnham[6] show that most reactions occurring in early infancy are due to foods. Block[7] has proven that substances causing allergic reactions are not necessarily protein in nature. White[8] has shown a definite connection between many cases of pylorospasm and eczema. It is to Vaughn[9] that we owe the theory of allergic equilibrium with its explanation of threshold values and quantitative reactions.

Many of the old explanations of the causes of eczema have been properly placed in the roles of exciting factors, thus assuming places of major importance without explaining the whole phenomena.

Racial and Social Tendencies.—Balyeat[10] states that eczema rarely occurs in the American Indian. In the examination of many hundreds of negro infants in the outpatient department of Grady Hospital I have observed eczema with comparative infrequency. Along with other allergic symptoms, it undoubtedly occurs more frequently as we go up the economic scale. The high strung, intelligent type of individual is more likely to be affected.

Family Tendencies.—The importance of the hereditary factor can hardly be overestimated. Balyeat[10] reports positive family histories of allergy in 76 per cent of a series of 181 cases of eczema. In my series of 83 cases, 74 per cent had positive family histories, and in the more intelligent group of private patients the percentage rises to 90.

Incidence of Eczema.—Cooke and Vander Veer[11] estimate the incidence of allergic reactions in the population as a whole at 10 per cent. The table below shows the incidence in a group of infants and small children recently examined in the health centers of Atlanta.

TABLE 1

Number of children examined	989
Children with eczema	60 or 6 per cent

In comparison, Yampolsky[12] reports an incidence of 2 per cent in a group of 500 infants examined in the Central Presbyterian Clinic of Atlanta. These children were drawn from the poorest sections of the city. The above figures would seem to bear out a previous statement that eczema is more frequent as we ascend the economic and social scale. It should be remembered, however, that many true allergic eczemas are mild and will disappear spontaneously as the child develops his own immunity. It is only the severe and persistent cases which demand further study.

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*Read in Section on Pediatrics, Southern Medical Association, Twenty-Fifth Annual Meeting, New Orleans, Louisiana, November 18-20, 1931.

*From the Department of Pediatrics, Emory University School of Medicine.

*The Fulton County Medical Society, Atlanta, awards each year the Fischer Prize for the best piece of research work by one of its members and this paper won that prize for 1931.

Fig. 1
The usual type of facial eczema.

Differential Diagnosis.—Many of the common skin diseases of infants and children may be confused with eczema. Perhaps the most commonly mistaken is the so-called acid rash, which means nothing. Infectious seborrhea, impetigo, scabies, ringworm, pyodermia, and the common diaper rash are often mistaken for eczema, and *vice versa*. Impetigo is a common complication of eczema as a result of scratching. Time does not permit a detailed discussion of each disease mentioned above.

Causes of Eczema.—The causes may be placed in two large groups: exogenous causes (occurring in adult life); and endogenous causes (occurring in infancy and childhood). The exogenous causes will be omitted from this discussion.

The endogenous causes may properly include foods, which are of greatest importance, and those conditions termed exciting factors, such as irritation of the skin, thermal changes, focal infections, constipation, and thyroid dysfunction. Of the foods the most important are listed in *Table 2*.

TABLE 2

Endogenous Causes	No. Cases
Cow's milk	30
Eggs	24
Wheat	13

Other reactions, in this series, of importance in one or more cases were caused by apples, grapes, grapefruit, tomatoes, lemons, oranges, plums, barley, corn, oats, rice, potatoes, beans, okra, pecans, beef, lettuce, fish, chocolate, pollen and bacteria.

The above table is quite similar to that reported recently by Smyth *et al.*[13]

Location of Eczema.—Eczema occurs most frequently on the cheeks, the forehead, the scalp, the flexor surfaces of the extremities, and in some instances over the entire body.

Complications of Eczema.—Impetigo has been the most frequent complication in this series. Seborrhea occurs frequently and scabies and pyodermia are not rare.

Laboratory Findings.—No known laboratory work is diagnostic. The lesions are essentially inflammatory in type. Eosinophilia is perhaps

Fig. 2 (top). Papular type of eczema.
Fig. 3 (bottom). Peri-anal eczema occurring in patient whose face is shown in Fig. 2. An unusual type. Duration, four months. Cleared in six days after diet elimination.

the most constant finding of importance, though it occurs in many other conditions.

DIAGNOSIS

Diagnosis may be considered under four heads:

Fig. 4 (top). First step in scratch testing.
Fig. 5 (center). The second step in scratch testing, application of the solvent.
Fig. 6 (bottom). Third step in scratch testing, the application of the powdered extract to form a paste over the scratch.

Fig. 7
Similar to Fig. 1

(1) Examination of the patient for the characteristic skin lesions which are primarily inflammatory in type and become dry and scaly as the lesions become chronic.

(2) (a) The family history may be studied in the possibility that it may reveal hereditary tendencies. (b) Detailed personal history may give valuable information as to the causative factors in the case. For instance, if the eczema appears soon after cereals are added to the diet, they are probably to blame.

(3) Testing (discussed in detail below).

(4) Diet elimination tests of Rowe,[14] Dale and Thornburg,[15] and Waters and Hopkins[16] are of value particularly in hospitals where the utmost in cooperation can be secured. They are also of value in cases where other testing is unsatisfactory for any reason. I have not found them necessary in working out the etiological factors in this series.

Testing.—There are four well known methods of testing:

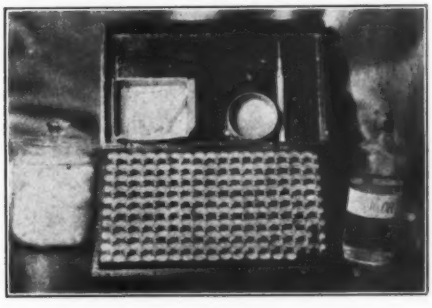

Fig. 8
Material necessary for scratch testing.

(1) The scratch test is the simplest and, because of the factors mentioned above, the most practical in infants. Material consists of an instrument for scratching (an ordinary needle in a handle), powdered extracts or allergens which are supplied commercially, and a solution of tenth normal sodium hydroxide. The allergens are applied in a thin paste over the scratches with flat tooth picks, one end of which is used to apply the powder and the other to apply the solvent.

(2) The intradermal method is more accurate than the scratch, or perhaps it is better to say that the latter is more sensitive. But it is far more difficult in children's cases and for that reason less practicable than the scratch. It may be used to check doubtful reactions.

(3) The patch test is more applicable to the exogenous eczemas of later life, but of occasional value with children.

(4) In the method of Walzer-Prausnitz-Kustner, or passive transfer, the serum of a sensitive individual may be used to sensitize local areas of skin in a non-sensitive individual. This method can be used occasionally when it is not possible to test an infant in the usual manner. When it is possible thus to transfer sensitivity to a non-sensitive individual the characteristic reaction is absolute proof of a true allergy.

The accurate interpretation of tests is of great importance and inaccurate interpretations explain many failures.

I have seen few urticarial wheals from the scratch system in eczemas. The most common reaction is a faint area of erythema around the scratch. Any elevation along the scratch or surrounding erythema greater than its neighbor should be regarded as a positive reaction. The erythema of irritation following the scratch seems to confuse some workers. It subsides in a few minutes, leaving the true positive plainly evident. Alexander and McConnell[17] have shown that the skin of the trunk is more sensitive to testing than the skin of the extremities. Vaughn[9] and others report a delayed reaction occurring at the end of twenty-four hours. I have not observed this, although I have often sought it.

TREATMENT

The effective treatment consists in the elimination of the offending foods. It is often necessary to substitute some other food for milk. In mild cases simply heating the milk or the use of a powdered lactic acid milk suffices. Where it is necessary to avoid milk, even in minute quantities, I have found soy bean milk to be of the greatest value. I have many patients who have thrived on it for many months. Its taste is a handicap, but small infants do not seem to object; many others learn to like it if they are continually offered it as food. In two instances the barley flour which it contained caused reactions (eczema). This fault was overcome by the substitution of a soy bean milk (special) containing corn flour instead of the barley flour. This excellent food was introduced by Hill and Stuart.[18] A special heat treated milk proved unsatisfactory in two cases of severe eczema. The great advantage of this method is that after a few months prohibited foods may again be taken in gradually increasing quantities. It thus stimulates an immunity.

Local treatment was scrupulously avoided in this series for fear of confusing results.

Relief of itching was best obtained by the use of 0.2 per cent ephedrine in jelly. The broken skin apparently absorbs enough of the drug to cause a capillary constriction and relief of itching and weeping.

CASE REPORTS

Case 1.—A boy, aged six years, had suffered from a severe facial eczema every winter since he was one year of age. The eczema appeared when he had an acute coryza, especially when it was complicated by sinusitis. The lesions would disappear when the sinusitis cleared until the past year, when it persisted into the spring months. Testing revealed a sensitivity to milk, eggs, and wheat.

Elimination of those items from his diet resulted in an immediate clearing of the skin. After three months' abstinence, the foods mentioned were again introduced into the diet in gradually increasing quantities, so that he now takes them without trouble.

Infection, in this instance, seemed to be the exciting factor, causing a flare-up of the underlying allergic reaction.

Case 2.—A boy, aged three months, had a severe eczema affecting the whole body, but most marked on the face and scalp. It first appeared at the age of one month and had steadily grown worse. His diet consisted of sweetened condensed milk.

Testing revealed a sensitivity to cow's milk. He was placed on a diet of soy bean milk and he improved, but did not clear completely. Retesting revealed a marked sensitivity to barley flour, which is found in the soy bean milk. He was then placed on a diet of soy bean milk special, containing corn flour instead of barley flour. The eczema cleared rapidly, and now, eight months later, he is still taking the soy bean milk, is normal in weight and entirely clear of eczema.

Case 3.—A boy, aged eight months, had a severe facial eczema of seven months' duration. He was breast fed.

Testing revealed a sensitivity to wheat and eggs.

Those items were removed from the mother's diet and the eczema cleared within ten days.

Case 4.—A girl, aged five years, had frequent attacks of urticaria, a few patches of papular eczema, and occasional digestive disorders.

Testing revealed sensitivity to eggs, tomatoes and lettuce. Those foods were taken out of her diet and after six months were again introduced s'owly. She has been free from symptoms for three years.

TABLE 3

SUMMARY OF RESULTS

Condition		No. Cases	Per Cent
Completely recovered		67	81
Improved		5	6
Outcome unknown	9 cases		
	} Failures	11	13
Known failures	2 cases		
Total number studied		83	100

CONCLUSIONS

(1) Most infantile eczemas are allergic in origin.

(2) Prompt and lasting relief can usually be obtained by thorough testing and the elimination of offending foods.

(3) The phenomenon of immunity will result in recovery, without treatment, in the mild cases and in the more severe also, but a long period of suffering can be eliminated by the method outlined above.

(4) Cow's milk, eggs and wheat are the most common offenders, but many errors and failures will result if they are the only factors considered.

(5) Local treatment, combined with testing and elimination, is probably the method to be recommended.

(6) The high percentage of good results in this series may be explained by the comparatively small number studied. Checking and rechecking was done repeatedly to insure correct figures. The unusually good follow-up work done by the social service worker undoubtedly contributed largely to the excellent results.

(7) A study of infantile eczema from the standpoint of allergic reactions offers a logical method of procedure based on scientific facts and substantiated by extensive clinical experience other than my own.*

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*The author was assisted by members of the staff of the Central Presbyterian Clinic and Mrs. Ralph Noland, whose excellent follow-up work aided materially the success of the work.

DISCUSSION (*Abstract*)

Dr. B. G. Efron, New Orleans, La.—Allergy has come to play a more and more important part in medicine. The allergic basis of many conditions can no longer be ignored.

Dr. Bivings is fortunate in having such a large series of cases due to foods. In many cases of eczema, however, foods are not responsible. Contact substances must be considered and tested for. Very recently we determined flannel to be the cause of an eczema occurring in winter in an infant of 21 months. Skin tests in this case were entirely negative, but patch tests reproduced the rash. This case was kindly referred to me by Dr. von Meysenbug.

The term allergy calls up at once skin tests. This is erroneous. The skin tests are only one procedure in the study of allergic disease. This procedure is a laboratory test, and as such is subject to all the vagaries and inaccuracies of laboratory tests in general. The etiologic factors are determined by skin testing in only 50 per cent of cases. The history is most important, for without the history the skin tests are without value. Clinical trial and careful observation are of paramount importance. Very often prolonged observation, with minute attention to the interpretation of the clinical course, is essential before an etiologic diagnosis is determined. In many food cases it is necessary to resort to diet trials.

Dr. W. Ambrose McGee, Richmond, Va.—We have a large colored population in Richmond which is about one-third negro. I have seen both asthma and hayfever in the colored race, in the darkest colored negroes I have ever seen. I do not know whether they were full-blooded negroes or not, but they certainly appeared to be.

HERPETIC FEVER WITH STOMATITIS: REPORT AND DISCUSSION OF A CASE IN WHICH THE VIRUS WAS ISOLATED*

By JOHN B. YOUMANS, M.D.,
Nashville, Tenn.

Herpetic infections[1] have assumed a new importance since it was demonstrated that the disease is transmissible to animals[2] and is caused by a neurotrophic,[3] filterable virus belonging to the group which includes those of rabies and anterior poliomyelitis. Additional importance has resulted from the belief of certain writers, particularly Levaditi,[4] that this virus is also the cause of human epidemic encephalitis.

These discoveries are the more interesting because certain forms of herpes, such as the "cold sore" or fever blister, are very common and familiar to everyone, although rarer forms occur which are much less well known. Herpes simplex should be sharply distinguished from zona or zoster, often called herpes zoster, which is not transmissible to animals, and shows certain clinical differences, though morphologically the lesions of the two diseases are practically indistinguishable. The many clinical varieties of herpes simplex described and named are in part the result of our earlier ignorance of the etiology. They are also the result of a classification based not only on the location of the eruption (herpes labialis, facialis, progenitalis, *et cetera*) but on the primary or secondary nature of the infection. Secondary or symptomatic herpes occurs not only in association with a great many infectious diseases, intoxications and other pathological conditions, but with certain physiological disturbances as well, such as menstruation, fever, *et cetera*. Herpes may also occur as an idiopathic disease and in this form is frequently recurrent, returning at the same site at regular intervals for long periods.

Ordinarily herpes is a local disease only. There is described, however, an acute idiopathic generalized herpetic infection followed by a local eruption and known as herpetic fever.

Morton[5] was probably the first to associate the presence of constitutional symptoms with an idiopathic herpetic eruption, though he believed the condition to be non-infectious. A good description, under the term febricula, is given by Griesinger,[6] who considered it a mild or abortive form of the typhoid, typhus, plague group of fevers which at that time were still incompletely differentiated. Plessing,[7] however, held it to be a separate and distinct disease with a specific etiology. A similar view has been held by many modern writers, though some have denied the existence of such an entity.[8] In addition to sporadic cases, epidemics of the disease have been described.[9] Some writers believe that a generalized herpetic infection may occur without a local eruption or with atypical lesions,[10] the so-called herpetic fever sine herpes. The occurrence of atypical lesions under certain conditions in the experimental transmission of local herpes to man gives some support for this belief.[11]

The disease, which most often occurs in young, healthy persons, begins suddenly, often with a chill, followed by a rapidly rising temperature. The patient complains of headache, fatigue, and pains in the back and extremities. Examination fails to show any cause for the symptoms. After two to four days of fever the temperature falls abruptly, often after a heavy sweat. Toward the close or shortly after the febrile period an herpetic eruption suddenly appears, most often about the face, but rarely confined to the mucous membrane of the mouth or throat. In many cases there is an enlargement of the regional lymph nodes and occasionally the spleen is enlarged. Recovery is rapid and uncomplicated by any other disease. Variations from these typical cases are seen.[12] The disease is of particular interest, not only because of its rarity,[13] but because it presents an opportunity to study an apparently generalized infection with the herpetic virus in man, uncomplicated by the presence of other disease.

CASE REPORT

Miss O. N., a student, aged 24, was first seen on May 19, 1931, complaining of a sore mouth. The illness had begun three days before when, on eating, she noticed that the roof of her mouth felt scalded, sore, and burned. She did not examine her mouth at this time. The following morning she felt fatigued, and was drowsy all day. In the evening she had a moderately severe bitemporal headache, felt hot and feverish, and a "sore" was noticed on her tongue. The following day the soreness of the mouth continued, the gums became tender, and a "sore" was noted in the corner of the mouth. The glands under the jaw became swollen and tender, and she felt feverish and "chilly." The next day, the third after the onset of symptoms, the roof of the mouth, the gums and the lips were red, swollen, and very tender. The glands under the jaw were larger and more tender. There was a generalized aching in the back with radiating

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pains down the legs to the knee. There were no other symptoms except constipation. She did not believe she had ever had a real fever blister, but was probably exposed to an herpetic infection two days before the onset. She occasionally had "canker" sores. Six weeks before she had been treated by me for a catarrhal jaundice, but had completely recovered three or four weeks before the onset of the present illness. The remainder of the history was unessential.

Examination.—The patient was unusually well developed and nourished. The temperature was 103° F.; the pulse rate 100. The skin was hot and dry, but was otherwise clear. The face was flushed, the eyes suffused and the conjunctivae slightly inflamed. The sclera were not jaundiced. The lips were swollen and red and in the right corner of the mouth, at the mucocutaneous border, there was a small whitish pustule with a red, inflamed base. The mucous membrane of the mouth and throat, except the palate, was red and swollen. The mucous membrane of the hard palate presented a whitish, scalded appearance with marked exaggeration of the rugae. The tongue was covered with a thick brownish-white coat. Scattered over parts of the gums and buccal mucosa was a thin, white membrane or exudate, which could be readily wiped away, leaving no ulceration. On the left side of the soft palate there was a shallow ulcer about a millimeter in diameter, covered with a grayish exudate and surrounded by a red, hyperemic zone. In the same region there was a whitish vesicle of about the same size. On the right tip of the tongue were a number of small, shallow ulcers similar in size and appearance to that observed on the soft palate. The regional lymph nodes, particularly the submaxillary, were swollen and painful. The epitrochlear lymph nodes, also, were enlarged and tender. The heart and lungs were normal. There was slight tenderness in the right lower quadrant of the abdomen. The spleen and liver were not enlarged. The remainder of the examination was essentially normal.

Because of the apparent idiopathic nature of the disease, the marked constitutional symptoms and the character of the lesions in the mouth, it was immediately suspected that she was suffering from one of the specific infectious diseases characterized by an ulcerative stomatitis, such as, for example, foot and mouth disease. Smears taken at this time from the lesions in the mouth showed only rare and atypical Vincent's organisms, thus excluding a more common cause of ulcerative stomatitis.

Course.—The following day the patient was admitted to the hospital with the temperature still in the neighborhood of 101° F. In the mouth three small ulcers, similar to those previously noted, were seen on the buccal mucosa just inside of the mouth on the right. The urine was normal except for the presence of red and white blood cells (menstruating). The white cells were 10,450, with 72.5 per cent polymorphonuclears, 0.5 per cent basophils, 24 per cent lymphocytes, and 3 per cent mononuclears; otherwise the blood was normal. The Wassermann reaction of the blood was negative.

The next day the temperature was still higher (102.2° F). Small ulcers similar to those described, appeared on the left side of the tongue, soft palate and upper poles of the tonsils. It was observed that she slept most of the time and she herself spoke of it and complained of being very sleepy. On the second day

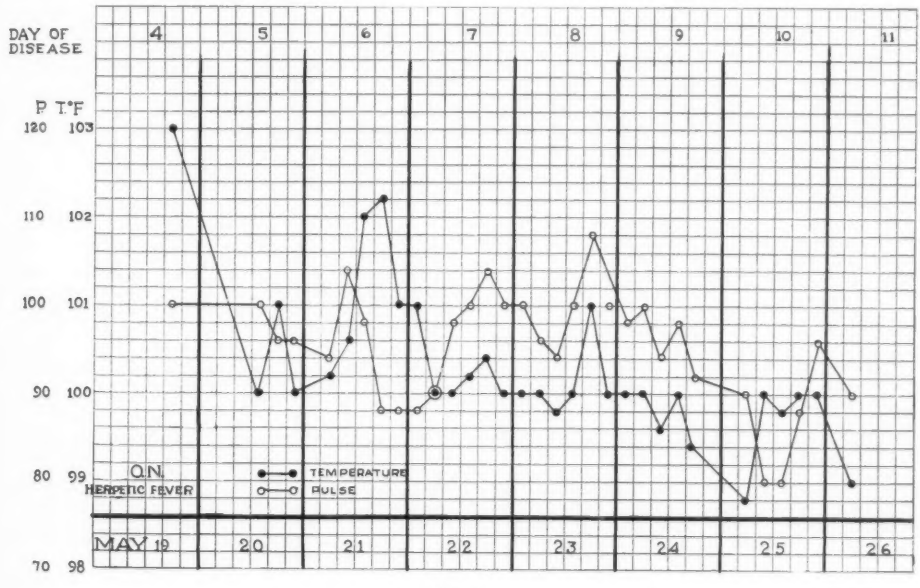

Fig. 1
Pulse and temperature curve during the course of herpetic fever.

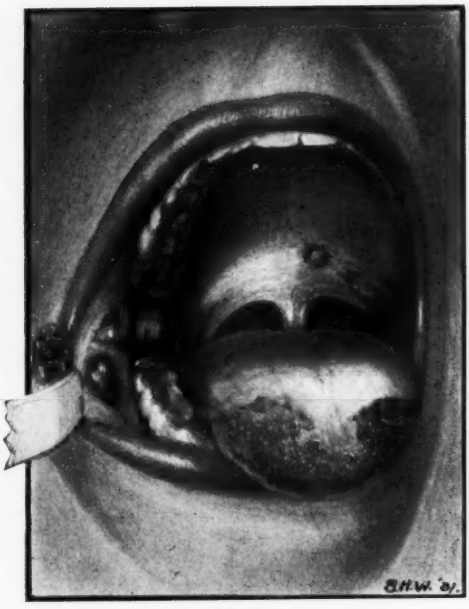

Fig. 2

A drawing of the mouth made on the sixth day of the disease. The individual lesions are shown well, but the swelling and redness of the mucous membrane of the entire mouth and the lips are not apparent.

after admission (May 22) a few small, scattered, reddish papules were noticed on the forearms, especially on the flexor surfaces. For the next two days the temperature remained between 100 and 101°, but her general condition improved. The leucocytes fell to 7,100, with a decrease in the polymorphonuclear neutrophils to 58 per cent. A lumbar puncture was done on May 23 and clear fluid under normal pressure was removed. There were 4 cells per cu. mm. and no increase in proteins. The mastic curve and Wassermann reactions were negative. By the fourth day after admission (seven days after onset) the specific lesions in the mouth had begun to improve, but there was some ulceration of the gums and now smears showed many Vincent's organisms. She was discharged six days after admission and two days after discharge the specific lesions in the mouth were entirely healed and the mouth was nearly normal.

Special Laboratory Studies.—In the meantime, with the cooperation of the Department of Pathology, the following special studies were made. The scarified cornea of a rabbit was inoculated with material obtained from the lesions in the mouth. Three days later there was a mild injection of the cornea with several small vesicles, and a typical herpetic keratitis soon developed. Six days after inoculation the animal showed constant rotation of the head to the right and the temperature was 105.5° F. The next day several convulsions were noted, but by the fifteenth day all symptoms had disappeared and the rabbit was killed for histological study. A second rabbit was injected intracerebrally with 0.5 c. c. of whole blood obtained on May 22 with entirely negative results. Spinal fluid obtained May 23 was injected intracranially into two rabbits: 0.25 c. c. of unconcentrated fluid in one, and 0.5 c. c. of spinal fluid concentrated by evaporation *in vacuo* to one-fifth volume in the second. Both remained well. Material obtained from the skin lesion in the right corner of the mouth was inoculated on the scarified cornea of a fifth rabbit. This animal developed typical keratitis on the third day and signs of encephalitis on the seventh, from which it had recovered by the thirteenth day, when it was killed for histological study of the cornea. Material from the lesions in the mouth injected into the foot of a guinea pig did not cause lesions of foot and mouth disease.

DISCUSSION

While the case described above is clearly one of idiopathic herpetic stomatitis, the question arises whether it is also one of herpetic fever. Before the discovery of the virus of herpes, the only evidences of the existence of herpetic fever as a clinical entity were the clinical features of the disease and its resemblance to other eruptive fevers. In the present case, the constitutional symptoms plus the isolation of the virus from the mouth, and the absence of other disease, seem sufficient to warrant a diagnosis of idiopathic herpetic fever. As far as we have been able to determine, isolation of the virus in such a case has not been previously reported.

These cases of herpetic fever are of particular importance with respect to the pathogenesis of herpetic infections. Much is yet unknown regarding the behavior of the virus after its entrance into the body. It seems probable that a dissemination of the virus often occurs, but the extent and the mechanism are uncertain. With the discovery of the etiologic agent it might be expected that a generalized infection could be demonstrated in these cases by finding the virus, not only in the local lesions, but perhaps in the blood and other body fluids. So far these expectations have not been fulfilled. Bastai and Busacca[14] have reported finding the virus in the blood and spinal fluid of many patients with herpes, and they believe that even in local herpes there is a general infection with a local eruption. Their results have not been confirmed and their later recovery of the virus from the blood and spinal fluid of a large percentage of normal persons,[15] also unconfirmed, has cast a doubt on their results in patients with herpes. Recovery of the virus from the blood of animals infected experimentally is difficult, though

local herpes may be produced by intravenous injections, particularly if combined with local injury to some part of the body. The available evidence indicates that the growth and development of the virus is of necessity intracellular. It is, therefore, unlikely that the virus is present, except accidentally and passively, in the blood stream. The failure to isolate the virus from the blood in the present case is of special significance because the general and constitutional symptoms of the infection suggests a widespread distribution. Most studies have been made on cases of local herpes.

One possible explanation for the failure to isolate the virus from the blood should be pointed out. The distribution of the virus may take place either before or after the development of the local eruption. If the distribution is secondary, the virus should be present in the blood, if at all, after the appearance of the herpetic eruption, a time when most attempts to isolate it have failed. On the other hand, if the distribution is primary, as seems more likely clinically, the virus would probably be present in the blood only during the short period between the onset of symptoms and the appearance of the local eruption. Inasmuch as the diagnosis of herpetic fever is rarely suspected before the eruption has appeared, blood studies will seldom be made until then, and until the virus has presumably left the circulation. Only in epidemics would it be likely that the diagnosis could be anticipated and proper studies made. No such studies have been reported. Therefore, the negative results in the present case, or in others, are not conclusive evidence that a distribution cannot occur by way of the blood stream.

In the present case it may be thought that the local symptoms and lesions present at the onset of the illness indicate a general distribution of the virus secondary to the local eruption. It is probable, however, that the characteristic lesions of herpes were not present at the onset and that the symptoms and local changes in the mouth were the result of an early injury to the cells during the period of general invasion before the development of the full-blown eruption. They may have been the result of localization in the mucous membrane, a region where such lesions and symptoms are more easily produced. It may be significant that in cases of herpetic fever in which the local eruption occurs in the throat, pain, soreness and swelling may occur during the period of constitutional symptoms and before the appearance of the typical eruption.

If the virus is not carried by the blood stream what mechanism is responsible for its distribution? Goodpasture and Teague[16] have shown that in rabbits the virus may pass from the site of inoculation at the periphery to the central nervous system by way of the nerve axis-cylinder. They have also shown experimentally that the virus may pass both centripetally and centrifugally. Thus it is possible for the virus, after entering the body, to invade a nerve, pass along it to the cord or brain, and from there proceed in other nerves to the periphery. With such a distribution the constitutional symptoms might be assumed to occur in association with the spread of the virus through the nervous system before the development of a local lesion either at the portal of entry or in the area involved by the efferent passage of the virus. Furthermore, a distribution by this means might follow either an exogenous infection, or a lighting up of latent virus in a peripheral nerve or ganglion.

Objection to the idea of a neurogenic distribution may be made because of the absence of central nervous system involvement in man and the failure to demonstrate the virus in the spinal fluid. The absence of central nervous system involvement is, I believe, more apparent than real. Most observations have been made on cases of local herpes. In herpetic fever, in which one might expect the widest distribution of the virus, there is good evidence of involvement of the central nervous system. Headache, dizziness, vomiting, nerve root pain, drowsiness, delirium, convulsions, stiff neck, and a positive Kernig's sign have all been observed. In fact, a meningeal type of herpetic fever has been described.[17] Philibert[18] has reported a case of severe genital herpes, appearing a week after herpes labialis, with high fever, stiff neck, headache, rigid spine, and a positive Kernig's sign. There were 13 cells in the spinal fluid and an increase in protein. Recently Pette[23] reported a case of repeated attacks of facial herpes preceded by fever, convulsions, unconsciousness, stiff neck, opisthotonos, positive Kernig's sign, and increased cells and protein in the spinal fluid. The virus could not be isolated from the blood or spinal fluid. Somewhat similar findings are reported by Szymanowski and Zylerblast - Zand,[19] Hirsch,[17] and Ravaut and Darre.[20] Even with

local herpes there may be evidence of involvement of the central nervous system. There is a type of genital herpes known as the neuralgic herpes of Mauriac, characterized by severe neuralgic pain about the ano-genital region, or even over a larger area, with paraesthesia, hyperesthesia, tenesmus and at times constitutional symptoms sufficient to warrant a diagnosis of herpetic fever. Ravaut and Darre[20] found an increased number of cells in the spinal fluid in 21 of 26 patients with genital herpes, and occasionally an increase in protein. In the case reported here the only evidence of involvement of the central nervous system was the headache, persistent drowsiness[21] and pains down the legs. However, it will be recalled that the virus was only mildly neurotropic and in interpreting the evidence of involvement of the central nervous system in any given case, the degree of neurotropism of the virus must be taken into consideration. In any event, the mere failure of the virus to cause severe brain and cord lesions comparable to those seen in rabbits does not preclude the possibility that a less severe and transient injury can occur in man. Even in rabbits the neurotropic virulence varies. In fact, such a variation in virulence may play a part in determining whether or not a given infection will result in a widespread distribution of the virus, an herpetic fever, or merely a local herpetic eruption.

The attempts to isolate the virus from the spinal fluid failed, as is generally the case. Except for the positive results of Bastai and Busacca referred to above, the virus has only occasionally been found in the spinal fluid and then as often in non-herpetics as in those with the disease.[22] This is not proof, however, that the central nervous system was not invaded. Perhaps the fact that the spinal fluid was obtained late in the disease is significant. More important is the finding of Goodpasture and Teague[16] that the virus is insulated in its passage up the nerve axis-cylinder and may penetrate deep into the cord or brain without causing lesions in the surrounding parenchyma. In rabbits developing encephalitis after peripheral inoculation the virus may not be obtained from the spinal fluid, and its presence there is usually the result of an intracerebral or subdural inoculation or an overwhelming, fatal encephalitis. Therefore, it would seem possible for the virus to invade the central nervous system in herpetic fever without being present in the spinal fluid.

SUMMARY

A case of idiopathic herpetic fever with stomatitis, in which the virus was isolated from lesions of the mouth, is reported. Attempts to isolate the virus from the blood and spinal fluid failed. The significance of the clinical features and laboratory studies with respect to the pathogenesis of herpetic fever is discussed.

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DISCUSSION (*Abstract*)

Dr. Ernest W. Goodpasture, Nashville, Tenn.—It is almost inconceivable that the common fever blister which has always presented evidences of its contagiousness was not recognized as infectious until ten or twelve years ago, when this was discovered by an ophthalmologist. Many laboratory studies have since been made with the virus, but practically nothing on the clinical side except to associate the herpetic virus with encephalitis as a possible etiological agent. This, I think, is the first report in which virus has been obtained from the lesion of herpetic stomatitis. The herpes virus is important, because it belongs to that neurotropic group of filterable viruses of which rabies, poliomyelitis and Borna disease of horses belong.

Two years ago there was reported before this Association a paper on the virus of yellow-fever. At that time I expressed myself as skeptical that the yellow-fever virus, although filterable, warranted grouping with the cytotropic viruses, meaning those viruses which evidence strongly indicates grow inside of the cell instead of outside of the cell. There is such evidence in the case of rabies, poliomyelitis, Borna disease of horses and especially in the case of herpetic infection in rabbits.

In the past two years it has been shown that the yellow-fever virus, which is infectious for monkeys, will induce an encephalitis in the mouse. When the virus from an infected mouse brain is passed through several generations, it will kill a mouse in six or seven days with an acute encephalitis; and then when the mouse brain is inoculated into a monkey brain the monkey will die of an acute encephalitis. In the latter case there will be no lesions in the liver and no other evidence of yellow-fever as it is usually indicated. The virus is still infectious for the monkey, but only for the central nervous system. In other words, the passage of the yellow-fever virus through the brain of a mouse so changes it that it attacks the nervous tissues exclusively. Thus the evidence that was not present two years ago in regard to the cytotropism of yellow-fever virus is now complete, because it has been shown that the virus will increase in quantity locally after inoculation into a suitable tissue, that is, the brain tissue. It had not been shown up to that time that the virus increased in the liver where characteristic cellular changes occurred. So at the present time, instead of four neurotrophic viruses, we must include a fifth, namely, that of yellow-fever after it has passed through the brain of a mouse.

So we are rapidly gaining a great deal of information about these viruses, about the susceptibility of their hosts, and about the way they operate in infected animals.

Now, the herpetic virus belongs not only with the cytotropic viruses, but to that specific group, the neurocytotropic viruses. Various viruses show different trophisms, that is, they exhibit a selectivity for certain cells in association with which they will grow. The neurocytotropic viruses grow best in the nervous tissue.

In the disease clinically known as herpetic fever which has been discussed by Dr. Youmans, it is a question whether the virus is disseminated through the blood stream or through the central nervous system or whether there is a dissemination at all from local lesions in the mouth. I think there should not be a confusion between herpetic fever and a generalized herpetic infection. Dr. Youmans, I think, did not distinguish those two things quite carefully enough. I rather gathered that his definition of herpetic fever would be a disseminated infection with the herpetic virus. This I do not think is necessary, nor is there sufficient ground for assuming that this is the case. I think that a fever associated with herpetic infection may be due to absorption of poisons from local lesions, without assuming a generalized infection. However, if a generalized infection should occur, there is the best of evidence for believing that the virus might become disseminated through the nervous system and not through the blood stream. Virus in a rabbit that dies from herpetic encephalitis cannot be recovered from the blood stream; it cannot be recovered from the cerebrospinal fluid except where there is a meningitis and absorption of the virus through the meninges. So there is no indication, theoretical or otherwise, that virus is readily disseminated through the blood stream or the fluids of the body. It certainly does not proliferate in the fluids of the body, and it differs in that respect from most bacterial invasions. It is an intracellular affair; and when it spreads, it spreads along the cells from the periphery to the central nervous system. When it reaches the central nervous system it may spread centrifugally from the brain down through the cord, along the axis cylinders, through the spinal process; it may spread from the cord out to the periphery within nervous tissue.

There are a good many other clinical manifestations associated with herpetic infection which should be investigated both from the clinical and from the laboratory side. For example, the question of whether the herpes simplex virus can cause a zosteriform eruption. There is some indication that herpes virus may be responsible for some cases classified as herpes zoster. There is still the question of herpes virus as a factor in epidemic encephalitis. Then there is the question of herpetic fever, about which we know nothing at the present time. We know nothing of the reasons for the association of herpes infection with lobar pneumonia, with meningitis, with malaria, and with certain other diseases. They all offer possibilities for a further attack upon the question.

Dr. Youmans (closing).—I think I have misled Dr. Goodpasture by my use of the term "generalized infection." I employed it, as far as the present case is concerned, in the clinical sense of an infection with general or constitutional symptoms, without, necessarily, an invasion of the whole body by the infecting agent such as occurs in septicemia. Nevertheless, it seems possible that some distribution of the virus may occur, perhaps by way of the nervous system as was suggested.

SOLITARY CYSTS OF THE KIDNEY*

By ROBERT W. MCKAY, M.D.,
Charlotte, N. C.

Solitary cysts of the kidney are seldom clinically recognized. They are usually found at post mortem examinations or as a result of kidney operations done with a diagnosis of renal neoplasm. I have been able to find only 256 cases of this condition in the medical literature. Of these, by far the greater number were found by the pathologist and not by the surgeon. They were formerly thought to be a form of the disease known as congenital polycystic kidney, but on close study the two pathological conditions are seen to be quite different and there is no foundation for the belief that they are pathologically related.

Classification.—Usually they are classified with reference to the contents of the cyst cavity into serous, those containing clear fluid, and hemorrhagic, those containing blood or its decomposition products. Hemorrhages which occur in large hypernephroma and which subsequently undergo encapsulation and autolysis should not be included, neither should the small subcapsular cysts of chronic nephritis. They may be unilocular or multilocular, consisting of only one large cavity or a number of small partitions. Many theories have arisen as to the formation of hemorrhagic cysts. Some writers state that they occur as a result of bleeding into serous cysts, others contend that they are encapsulated hematomata or that the cysts originally consisted of a neoplasm which has been thinned out and destroyed as a result of the expansion from within. The latter viewpoint probably arises from the common finding of portions of malignant neoplasms in the walls of hemorrhagic cysts. Judd and Simon have reported two hemorrhagic cysts which they believe originally were aneurysms because of the finding of elastic tissue, intima and endothelium in the cyst walls. They advanced the theory that hemorrhagic cysts arise originally as renal aneurysms. We do not include in this discussion the large subcapsular calcified cysts which result from severe injury to the kidney. These subcapsular cysts are originally subcapsular hematomata which undergo autolysis and calcification of the cyst wall.

Etiology.—The three most common views held regarding their origin are: first, they are due to embryonic rests; second, they are due to failure of union between the glomeruli and the collecting tubules; and, third, they are retention cysts due to constriction of the tubules by fibrous tissue or blocking of the tubules by degenerated cells or detritus.

Hepler has recently brought forth a very interesting explanation of their origin. He thinks that the underlying causes of all large solitary cysts of the kidney are the same, and has produced such cysts in rabbits' kidneys. His theory is that they all result from an interference with urinary drainage of a portion of the kidney plus a simultaneous interference with its blood supply, and that when we have these two factors present, namely: urinary obstruction plus ischemia, cysts are formed. If the underlying cause be

*Read in Section on Urology, Southern Medical Association, Twenty-Fifth Annual Meeting, New Orleans, Louisiana, November 18-20, 1931.

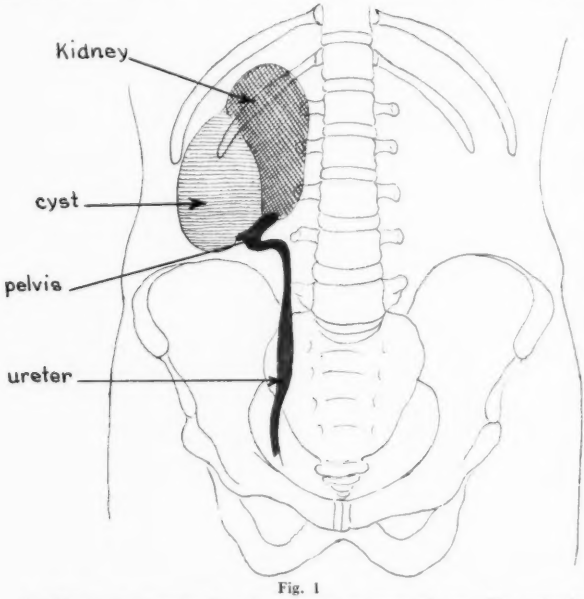

Fig. 1

A solitary cyst occurring in a boy twenty-one years of age. The patient's chief symptoms were discomfort in the right side and pus in the urine. Note that the pressure produced by the cyst on the renal pelvis creates a crescentic filling defect which is very suggestive of renal neoplasm. The outline of the cyst was plainly visible in a plain x-ray plate.

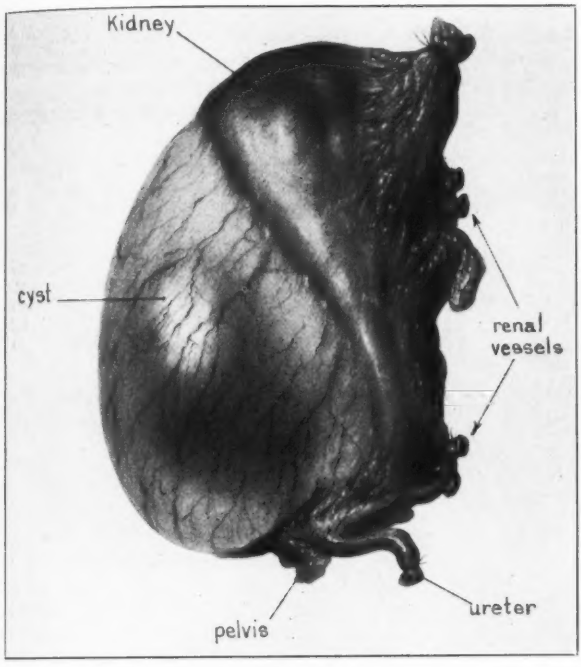

Fig. 2
Drawing of the kidney removed in Case 1. Note the double vascular pedicle, also the deformity of the uretero-pelvic junction and pelvis.

aneurysm or tumor, we may very well have as a result hemorrhagic cysts.

Recently the following two cases of solitary cysts of the kidney have come under our observation.

Case 1.—A white man of 21 gave a negative family history.

He presented a history of long-standing discomfort and pain in the right side of his back. He first experienced this pain when tackled in a football game about ten years previous to examination. He acquired lues, and during the course of his luetic treatment his family physician found pus in his urine and referred him to us for examination.

The patient was a well-nourished man of 21 years, who complained of definite pain and muscle spasm in the region of the right kidney. Manual palpation of the left kidney region did not reveal a mass.

Examination of the patient's genitalia and prostate were normal.

Cystoscopy revealed a slight degree of cystitis and trigonitis. The ureteral orifices appeared normal.

Ureteral catheterization was carried out and clear urine was obtained from both kidneys.

The right kidney urine showed a specific gravity of 1.010 and numerous pus cells. The left kidney showed a specific gravity of 1.014 and no pus cells.

The urine sediments from both kidneys were stained for tubercle bacilli, with negative results.

Intravenous 'phthalein injections showed 21 per cent excreted in the first one-half hour from the left kidney, and no dye from the right kidney in one-half hour.

X-Ray.—A flat plate was taken, which revealed the outline of a cystic mass in the region of the right kidney.

A bilateral pyelogram was then taken. Ten c. c. of sodium iodide were injected into the right kidney and 15 c. c. into the left. The pyelogram of the left side was normal in appearance. The right renal pelvis presented a definite flattened appearance with a crescentic filling defect. A diagnosis of solitary cyst of the kidney was made from the appearance of the cyst outline in the flat plate plus a crescentic filling defect. A nephrectomy was carried out quite easily and the patient made an uneventful convalescence.

The specimen proved to be a large solitary cyst of the kidney (*Fig. 1*).

The plain x-ray plate, showing the cyst outline, was more helpful in making the diagnosis than was the pyelogram.

Case 2.—A white woman, age 51, complained chiefly of pain in the region of the right kidney.

Her family history was irrelevant. Her past history was quite interesting. To her certain knowledge she had had discomfort in the region of her right kidney over a period of four years. The pain was rather indefinite, but was localized in the right upper quadrant. An exploratory operation had been done three years previously for a gall-bladder. At this time it was recognized that she had an enlarged kidney.

Since the patient's exploratory operation, she noticed

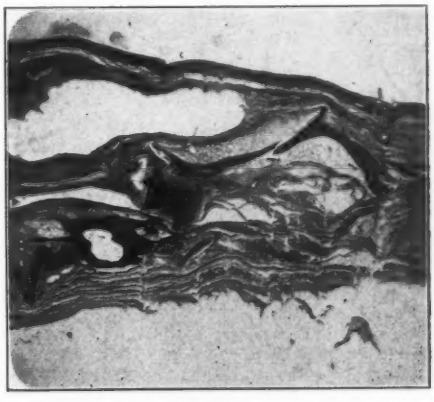

Fig. 3
A section of cyst wall.

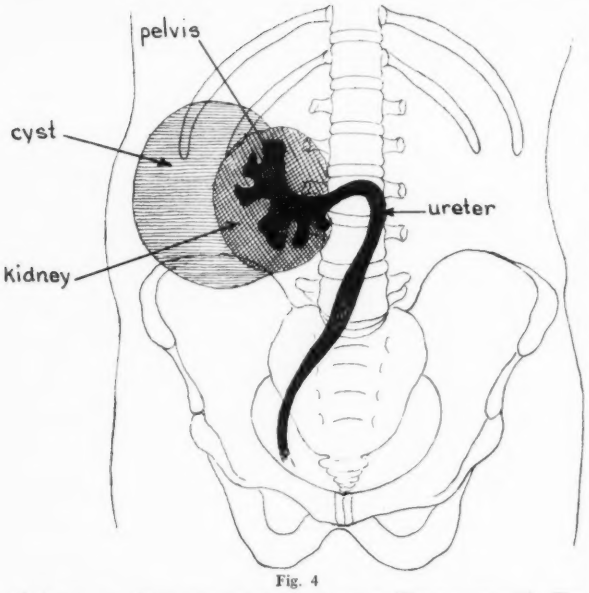

Fig. 4

Pyelogram of solitary cyst occurring in a woman fifty-one years old. The ureter was pushed over the vertebral column. The deformity of the pyelogram is quite different from the preceding case. It was erroneously thought to be a hypernephroma of the kidney.

that the mass in the region of her right kidney progressively grew larger. There was no acute pain, but rather a feeling of discomfort. There had been no chills and fever.

The patient was a very stout, over-nourished female of 51 years. The lips and mucous membrane were of good color. Although she had had the tumor in the region of her right kidney for four years, she had not lost any weight.

There was present in the right upper quadrant a freely movable mass about the size of a grape fruit. This mass was not adherent and could be moved to the midline quite easily. The surface of the tumor was quite smooth, and although there were no irregularities, it felt quite firm. Abdominal palpation was somewhat unsatisfactory, due to the fat abdominal wall.

The cystoscope revealed a normal trigone and bladder fundus.

No. 7 ureteral catheters ascended quite easily to the kidneys and clear urine was obtained from both sides.

The right kidney urine had a specific gravity of 1.032 and contained many pus cells. The left kidney showed a specific gravity of 1.031.

A pyelogram was taken (*Fig. 2*), which showed a large mass that apparently was in the lower pole of the kidney and had pushed the ureter well over the vertebral column. There was very little distortion of the renal pelvis and no tumor outline was perceptible. It was thought that the most probable cause of the displaced ureter was a hypernephroma of the lower pole.

A nephrectomy was carried out quite easily under spinal anesthesia.

The tumor proved to be a solitary cyst of the kidney (*Fig. 3*).

The patient made an uncomplicated recovery.

Manual examination of this patient was very difficult because of the great amount of abdominal fat. The history of having a tumor of the kidney for four years without symptoms or fixation should have made us suspicious of solitary cyst. The free motility of the kidney after the tumor had been present for four years should also have given us a clue as to its true nature.

Symptoms and Diagnosis.—Since large solitary cysts of the kidney do not communicate with the renal pelvis, they do not produce urinary symptoms save those which arise from pressure. It is possible for cysts arising in the upper pole of the kidney to produce symptoms by pressure on adjacent structures before they are palpable, but cysts of the lower pole are usually palpable and recognized as renal tumors rather early in their growth. Soli-

Fig. 5

Photograph showing the kidney flattened out on the surface of the cyst. Compare the size of the cyst with the size of the kidney. The kidney has been partially separated from the cyst wall by artifact.

tary cysts are usually confused with neoplasms of the kidney. Occasionally, however, they may be confused with blocked off congenital hydronephrosis or pyonephrosis.

Pyelography does not always differentiate them from neoplasms, as solitary cysts by pressure quite easily produce filling defects in the kidney pelvis. The kidney function may or may not be affected, depending upon how much kidney parenchyma has been destroyed. Exploratory puncture is mentioned only in order to condemn it as being unsurgical and dangerous. We think that after the colon is emptied thoroughly of gas with enemas, a good flat plate showing the kidney outline gives perhaps the best clue to the diagnosis of cysts. The two pyelograms of our cases show quite different appearances. In one the cyst has displaced the ureter over the vertebral column. In the second case it has merely flattened out the pelvis by pressure.

Treatment.—Solitary cysts of the kidney should routinely be operated upon, since it is impossible from urological examination to tell whether or not one is dealing with a serous or hemorrhagic cyst.

If at operation a serous cyst is found and sufficient renal parenchyma is left, a resection of the cyst should be done in the same way that one does a hemi-nephrectomy.

If the cyst be hemorrhagic, a nephrectomy

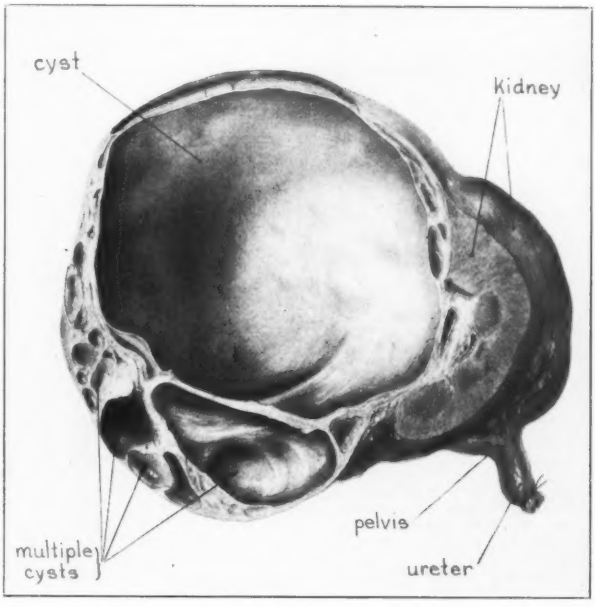

Fig. 6
A bisection of kidney and cyst wall of Case 2. Note the occurrence of the small cysts in the wall of the large cyst. The error in diagnosing this case was probably due to the patient's obesity.

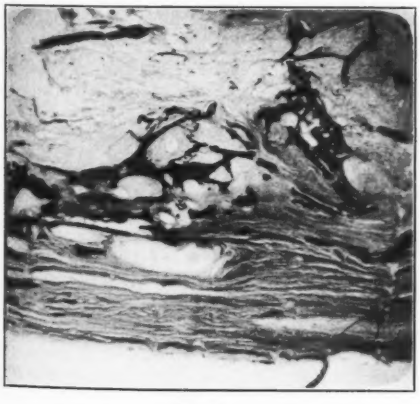

Fig. 7
A section taken of cyst wall of preceding case.

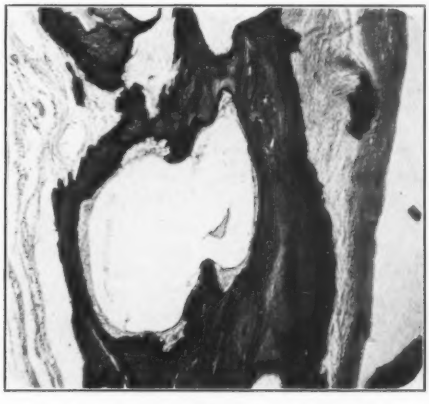

Fig. 8
One of the small cysts occurring in the wall of the large cyst. The dark area surrounding the cyst consists of calcium deposits.

should routinely be done. We have seen a hemorrhagic cyst in which a resection was done. When the specimen was examined a section through the cyst wall revealed a hypernephroma. The literature has a number of similar case reports. Therefore, we advocate, routinely, nephrectomy in hemorrhagic cysts.

CONCLUSION

Solitary cysts of the kidney are rare. We think that Hepler's explanation that they are the results of urinary obstruction to part of the kidney parenchyma plus interference with the blood supply is the most logical theory which has been advanced. A plain x-ray with the colon cleared of gas will usually show the cyst outline. Pyelograms may vary. They are usually confused with renal neoplasms. All solitary cysts of the kidney should be operated upon. The serous cysts should be resected where sufficient renal parenchyma remains. In all solitary cysts of the kidney that are hemorrhagic, however, a nephrectomy should be done.

Medical Arts Building

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DISCUSSION (*Abstract*)

Dr. Emmett B. Frazer, Mobile, Ala.—I should like to present an additional case of simple serous cyst of the kidney, proven by operation.

The prolonged history, presence of tumor in the renal area of regular outline and free mobility, absence of hematuria and disturbance of urinary function, negative cystoscopic findings and the pyelographic film gave sufficient information for a correct preoperative diagnosis.

It is quite easy to understand how small, simple cysts can be overlooked even in a careful investigation, inasmuch as they occur in the renal parenchyma and do not involve the pelvis of the kidney, and symptoms are also often quite indefinite. The effect of such a tumor is largely mechanical, and no pressure symptoms are noted until after it has attained considerable size.

Hepler's contribution to this subject is worthy of especial note. His theory of formation of the cyst is plausible.

Several years ago Dr. Charles Mayo called attention to the use of small pieces of muscle tissue upon which hemostatic mattress sutures were tied to prevent their cutting through after renal section had been done.

Dr. H. C. Gayden, Nashville, Tenn.—In no other phase of renal disease has there been so much speculation as to etiology, uncertainty as to pathogenesis, and lack of adequate classification as in the so-called solitary cyst of the kidney. The term "solitary" is used to distinguish this condition found in adult life from the congenital polycystic kidneys and from the multiple small retention cysts of chronic nephritis. Many large and even multiple cysts and bilateral cysts have been reported in the literature as solitary. Quite often hemorrhagic cysts have been included in the class of large solitary serous cyst.

From the study of various reports it is evident that all solitary cysts have not a common histogenesis. They may differ as to number, size, contents, sac wall, and associated renal disease. The direct cause may vary and with it the pathological features, but the mechanism of the cyst formation is essentially the same.

Alexander B. Hepler, writing on this subject and reporting 7 cases, 4 serous and 3 hemorrhagic, reviewed the literature and reported 216 serous and 40 hemorrhagic to date.

The increasing number of reported cases of solitary cysts indicates that the condition is not so rare as was formerly supposed. They are comparatively rare clinically, because many of them do not grow sufficiently large to cause symptoms, but with our newer methods of kidney examination we are going to see more and more diagnoses of conditions that would be hidden until late manifestations of incurable disease. Branch, a pathologist, reports that solitary cysts are occurring in 3 to 5 per cent of all autopsies. This being true, it behooves us to make a more careful study of our x-ray plates. Kretschmer says that it is possible to diagnose large solitary cysts in the great percentage of cases with the x-ray. Eisendrath says pyelograms will show a deformity of the pelvis or calices simulating that of a neoplasm. Now, with our intravenous urograms we shall no doubt be able to pick up early cases long before so much kidney destruction has taken place. The advantage of this is the comparison of bilateral pyelograms.

Caulk has studied a large series of cases and believes that those who contend that all kidney cysts are of congenital origin are unquestionably making a statement which is unjustified. Some found in infancy at autopsy would seem to have their origin in a disturbance during embryonic development, while the majority seem to belong to the class of retention cysts following obstruction. This later theory is borne out in the adult cyst in that 45 years is the average age incidence, a period when vascular lesions such as arteriosclerosis, aneurysms, infarcts and tumors of these cysts associated with lesions which do interfere with the blood supply.

Recently the production by Hepler of cyst formation experimentally similar in every detail to that found in the human kidney makes me lean toward the obstruction theory more than toward that of a malformation during fetal life. Eisendrath and Rolnic in their text-book classify solitary cysts as congenital. Eisendrath and Kretschmer and others state the location is more often at the lower pole of the kidney. The condition is found more frequently in women than in men.

There are no typical symptoms, but the most pronounced sign is a mass found upon routine examination of the abdomen at the lower pole of the kidney, for those at the upper pole cannot be felt unless the kidney is ptosed.

Treatment, of course, is excision of the entire cyst. This is possible if the cyst has not grown too large, destroying too much function or causing too large areas of dissection with the consequent risk from hemorrhage. Occasionally a cyst is located near the mid-portion of the kidney and separates the two poles so that it is impossible to dissect it out and rejoin the two poles and get much of a functioning kidney. Then I believe in these extreme cases nephrectomy is still the procedure of choice.

George F. Cahill, in the *American Journal of Surgery*, 1930, says that the usual recommendation of treatment is resection of the cyst. However, nephrectomy has been the procedure in most cases in spite of recommendation. He reports having operated and observed that in 14 cases all were nephrectomized except one, and it was resected successfully. Could these cases have had earlier diagnosis before the cyst pressed out much vital tissue of the kidney, resection could have been carried out and life prolonged.

Dr. J. A. C. Colston, Baltimore, Md.—I wish to call attention to a series of calcified cysts which we have seen in the last few years. As Dr. McKay has pointed out, these are not true cysts of the kidney. I think the history of these cases will show that they are the end result of traumatism with extravasation of blood from rupture of the kidney.

The first patient came complaining of pain on the left side. The plain roentgenogram showed a calcified cyst in the kidney region. We thought at once of a tuberculous process. The patient was incapacitated on account of pain in the lumbar region so that operation was advised and the kidney explored by Dr. Hugh H. Young and Dr. Dean Lewis. A calcified cyst was found adherent to the kidney and also so closely bound down to the peritoneum that it was evident that it could not be removed without endangering the blood supply of intraperitoneal viscera. The patient's symptoms still persisted after operation.

Another case, furnished us through the courtesy of Dr. Goldstein, presented an enormous mass in the abdomen and gave a history of having been run over by a wagon sixteen years before. The patient was severely injured, but a definite history of hematuria was not obtained. The enormous cystic mass in this case was completely excised, but the shock was so great that the patient succumbed.

We have had two other cases that presented the same picture of calcified cyst, each with a history of severe trauma in the region of the kidney. In one case the patient's disability was such that an operation was thought advisable. The cyst was exposed, but again could not be completely excised on account of dense peritoneal adhesions. As much as possible of the wall was excised, the remainder marsupialized and drained.

The lessons to be learned from this series of cases are obvious. When a symptomless calcified cyst is found it should be let alone. If sufficient symptoms are present to produce disability the cyst should be exposed, as much as possible of the wall removed, the remaining portion of the cyst collapsed and drainage instituted.

Another point which has direct bearing on Dr. Floyd's paper is the prevention of this condition, which may readily result in serious disability. Trauma to the kidney should not be neglected and when there is any doubt as to the presence of extravasation of blood or urine, prompt operation and evacuation of clots about the kidney should be carried out, as this series of cases clearly shows that if the extravasation be allowed to remain untreated a calcified cyst may result.

Dr. Arthur Sprenger, Peoria, Ill.—Dr. McKay's presentation recalls a case of solitary renal cyst reported in the *Illinois Medical Journal*, May, 1927, by Dr. J. R. Vonachen and myself.

The patient was a boy aged five years. A huge painless abdominal tumor was present and a diagnosis of malignant growth of the kidney was made. At operation a huge solitary cyst of the lower pole of the kidney was found. This was the sixth case of solitary renal cyst in children noted in the literature.

OLEOTHORAX: ITS USE IN CHRONIC NON-INFECTIOUS PLEURAL EFFUSION*

(REPORT OF TWO CASES)

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Oleothorax was first used in 1915 when Burnand[1] introduced 60 c. c. of gomenol and oil in a pyothorax. Fournier,[2] of Bourdeaux, in 1916 injected as much as 20 c. c. of gomenol and oil to cut short the course of a serofibrinous pleurisy. Wagner and Recklingausen[3] were the first to note that in the injection of oil into the peritoneal cavity there was an obliteration of the lymphatics which lasted over a considerable period of time. Bernou,[4] in 1921 and 1922, noted that there was a dual effect from the oil: first, the mechanical effect; and secondly, that

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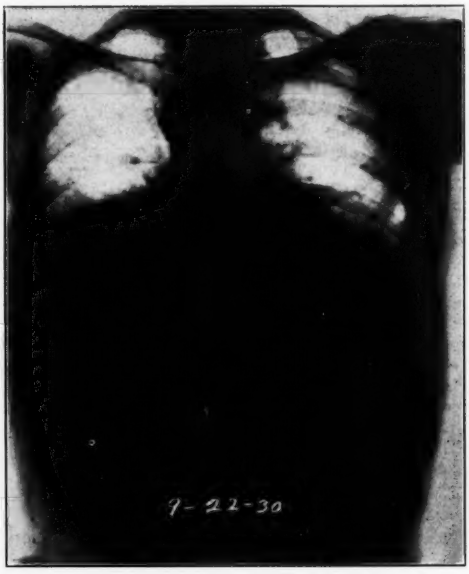

Fig. 1
Case 1. Case as originally seen with large bilateral effusion.

the gomenol and oil had a definitely antiseptic effect. Bernou, in 1918, had observed that 20 per cent gomenol and oil was well tolerated by the pleura, and used as much as 500 c. c. to prevent friction. However, Kuss,[5] in 1925, gave the first and best outline of the indications and technic of modern oleothorax.

At the present time oleothorax is indicated: (1) as a means of compression in cases unsuitable for pneumothorax; (2) to prevent recurrent effusion in artificial pneumothorax; (3) for the sterilization of infected pleurisy with effusion; (4) as a preventive against progressive adhesions; (5) in the healing of pleuro-pulmonary perforations; (6) to increase the rigidity of the mediastinum. The two oils that have been most commonly used are paraffin oil and olive oil. One case of mediastinal paraffinoma has been noted from the use of paraffin oil, as reported by Fontaine.[6] At the present time olive oil is most commonly used, to which gomenol has been added. Gomenol is the distillate from the leaves of the *Melaleuca viridiflora* and is non-irritating, but definitely bactericidal. It is used in dilutions of from 3 to 20 per cent, 5 per cent being most commonly used. Injections begin with 10 c. c. and if this is well tolerated the amount is rapidly increased every other day to the amount necessary for clinical results.

Experimental work on oleothorax was begun as early as 1922 by Archibald, who injected liquid paraffin oil into the pleura of animals with the production of a mild inflammatory reaction. Chandler and Gloyne[7] added gomenol and lipiodol to the liquid paraffin, pleural injections of which resulted in soft oleaginous adhesions, with early connective tissue, and new formed capillaries in adhesions of long standing. Ballon[8] confirmed these results in experimental work soon to be published. Waitz[9] injected the pleura of normal animals with olive oil containing oil of sesame, and sometimes 8 per cent gomenol. A fibrinous reaction was provoked in from twenty-four to forty-eight hours. He also found that the amount of oil was quickly absorbed and that the pleural fluid contained leucocytes and lymphocytes as well as fat droplets. Ballon,[8] in a small series of rabbits injected with paraffin oil containing gomenol and lipiodol, found no changes in the contralateral pleura.

In our series of guinea pigs and rabbits injected with 5 per cent gomenol in olive oil, we merely confirmed the findings of the cited experimental results. Our animals developed a

Fig. 2
Case 1. Chest immediately after bilateral thoracentesis.

Fig. 3
Case 1. One month after right oleothorax, showing markedly thickened pleura.

very definite pleural reaction with the formation of thin oleaginous adhesions. The severity of the pleural reaction was in proportion to the amount and strength of oil used; if small amounts were used first, and then gradually increased, the oil was much better tolerated, and the reaction less acute. There was definite absorption of the oil in all cases, but the amount varied greatly in the individual animals.

We realize that the reaction of virgin pleura cannot be readily compared to that of an infected and thickened one. But in both of the following cases the comparison is directly applicable, for in neither of these cases was there any sign of infectious reaction in the pleura.

The two cases we wish to report present a hitherto unemployed application of oleothorax.

Case 1.—The first patient was an elderly woman who, following a bronchopneumonia, developed a pleurisy with effusion and a myocardial insufficiency. She recovered from her acute illness, the heart became compensated, but the effusion, which at first was right-sided, persisted and soon became bilateral. Because of severe dyspnea, thoracentesis became necessary every few days. The fluid obtained was of low specific gravity, low in albumin content, and contained a few lymphocytes and polymorphonuclear cells. Repeated bacteriological studies and guinea pig inoculations were uniformly negative. The patient was completely incapacitated both physically and mentally because of the recurrent effusion and the frequent necessity of thoracentesis. We were in constant fear because of her debilitated condition and the necessity of the frequent tapping that at any time we might get a contaminating infection. In going over the literature we noted the use of gomenol and oil in the treatment of infected tuberculous effusion, and the reports not only stated that the purulent fluid was changed into a sterile simple transudate, but it also materially diminished the amount of fluid. Because of the hopelessness of the outlook in our patient we felt justified in applying this therapeutic measure to an entirely new field.

Following the technic of Kuss,[5] we withdrew approximately one-half of the fluid in the right side and slowly injected 10 c. c. of 3 per cent gomenol in olive oil. Two days later the same amount of fluid was withdrawn and 30 c. c. of oil were injected. At two day intervals we rapidly increased the amount until at the fifth injection we withdrew all of the emulsion possible and replaced it with 250 c. c. of oil. This was followed by a slight rise in temperature, leucocytosis, pleural pain, and a friction rub. The character of the fluid at this time was entirely changed. It was much thicker, with large amounts of ropy fibrin, and the leucocytes greatly outnumbered the lymphocytes. After one week the oil was withdrawn. Following this there were no signs of reaccumulation of fluid, but all of the characteristic findings of a marked pleural thickening. Curiously enough, the effusion on the opposite side also ceased, probably due to a continuity of inflammation, but six weeks later this untreated side again began to refill. The same routine was then applied to this side with identical results. At the present time, eight months after the injection of the first side and four months after the second, there has been no recurrence of fluid on either side. The physical signs are now those of a chronic pleural thickening.

Case 2.—The second case was also an elderly female

Fig. 4
Case 1. Marked pleural thickening one month after bilateral oleothorax.

who, two years previously, had had a radical breast operation by Dr. Harvey G. Mudd for Paget's disease of the nipple. There had been a recurrence in the skin, which was also operated upon shortly before she was seen. At examination it was found that she had a marked hypertension, cardiac hypertrophy and an interstitial nephritis. She was decompensated and during the course of her decompensation developed a right-sided pleural effusion which required tapping every four or five days. The fluid was a moderately heavy transudate, dark in color, rather high in albumin content, with a moderate number of lymphocytes, leucocytes and erythrocytes. Several attempts to find tumor cells by Goldman's[10] technic were negative. In spite of the return of compensation we continued to have a persistent right-sided effusion which had to be tapped at least once a week to relieve her dyspnea. Oil was injected exactly as in the first case with an identical result. After the last injection she developed an acute pleurisy and, following this, all the signs of a marked pleural thickening. A period of four months has now passed since the last injection of oil and there has been no recurrence of the fluid on the injected side. However, within the last two or three weeks an effusion has accumulated in the untreated side with a recurrence of severe dyspnea. Thirty-six ounces of this fluid were withdrawn and the stained smears showed definitely malignant cells. The original side into which the oil had been injected was tapped and only about 5 c. c. of oil emulsion were obtained. So that in the final analysis of this case we were dealing not with an effusion on a cardiac basis, but with one due to malignant metastases in the pleura.

The results in these two reported cases were most gratifying, because they relieved the patient not only of the distressing oppression of recurrent effusion, but also from the pain and apprehension of repeated aspiration and the possible danger of infection. The mechanism, however, that brings about the results obtained in these cases, is in apparent direct contrast to that which accomplishes the results in the treatment of tuberculous effusion as reported in the literature. Our results were obtained by the formation of an adhesive pleurisy with obliteration of the cavity, which prevented the reaccumulation of fluid. In tuberculous and infectious effusion the gomenol and oil is used to prevent such adhesions and permit collapse. Gomenol and oil has apparently a non-irritating effect in the presence of tubercle bacilli as shown by its toleration in the treatment of tuberculous cystitis. The non-irritating quality of gomenol and oil in the pleural cavity in the presence of tuberculosis is further suggested by the numerous reports of the resumption of artificial pneumothorax after the treatment with oil of infectious tuberculous pleurisy with effusion. The technic, however, is quite different in the two types of cases, and upon this difference we feel depends the difference in our results. In tuberculous pleurisy a portion of the effusion is withdrawn, then oil is injected and reinjected in large amounts, replacing the infected fluid with an oily emulsion of gradually increasing strength. In simple effusions such as existed in the reported cases as much of the fluid as is possible is withdrawn at each aspiration and the oil is reinjected so that each time the chest is tapped we have a marked increase in the strength of the emulsion and a diminution in the amount. In the infectious case the gomenol and oil acts as a disinfectant, a lubricant and a mechanical aid. In simple effusion it acts merely as a mildly antiseptic and somewhat irritating foreign body, with an obliterative pleurisy as the end result.

In conclusion, we feel that medicated oil in a simple serous effusion sets up an irritative pleurisy, which is followed by adhesions, with an obliteration of the pleural cavity. The mechanism bringing about these results is in direct contrast to that noted in the tuberculous and infectious pleurisies. We feel that the use of gomenol and oil is not only justified, but indicated, in cases of recurrent, mechanical, pleural effusions in which the indirect methods of medical treatment are ineffectual.

We acknowledge with pleasure our indebtedness to Dr. L. S. N. Walsh, Pathologist to St. Luke's Hospital, for his assistance and suggestions in carrying out the experimental work reported.

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Discussion follows paper of Dr. Ringer, page 246.

FACTORS CONCERNED IN THE PROGNOSIS OF PULMONARY TUBERCULOSIS*

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The appraisal of the condition of a patient presenting himself with pulmonary tuberculosis involves necessarily the making of a prognosis, for after the question: "What is the matter with me?" there follows its corollary: "What is my outlook?" It is this question of prognosis that I wish to discuss in some of its phases.

We have at our disposal five groups of observations upon which to base our conception of the case and, therefore, our prognostic attitude. These are:

(1) History
(2) The physical signs elicited upon examination
(3) The evidence furnished by the x-ray
(4) The general condition of the patient as revealed by (a) subjective and (b) objective symptoms
(5) The economic situation and the intelligence of the patient

History.—The history has by no means the same importance in prognosis that it has in diagnosis. A family tree heavily tainted with infection by tubercle must tend to a more guarded forecast. Duration of disease as obtained from the history, coupled with its intensity as derived from the same source, will influence the prognosis. Prolonged invasion with tubercle with a history denoting obvious fibrotic tendencies will naturally make for a better prognosis than prolonged invasion associated with necrosis and cavitation, or brief invasion pointing toward pneumonic or bronchopneumonic processes. Occasionally a patient will give a history of several near relatives who have had tuberculosis and who have uniformly recovered. In such a case, provided the history can be assumed to be reliable, these factors should be placed on the credit side of the prognostic ledger.

Physical Signs.—Physical signs are today just what they were in 1820, when Laennec conclusively described them. The signs have not changed, and Laennec's celebrated descriptions have yet to be improved upon. There has been much discussion as to what warrants a diagnosis of activity on the basis of physical signs alone; many papers have been written on the moist rale and the dry rale, but Lawrason Brown is eternally right when he states in his "Tuberculosis Theses:" "The most certain thing about the physical signs of 'activity' or 'softening' is their uncertainty."

Physical signs, moreover, being heard by the individual ear and conveying a certain impression to the individual intelligence are open to many different interpretations. The personal equation plays an enormous role in their evaluation, and the personal equation is not infrequently wrong. Consequently it is manifestly unfair to rely mainly upon physical signs for an estimate of the future progress or regress in the individual patient.

X-Ray.—The advent of the general use of stereoscopic x-ray films in the study of the existence and evolution of lung disease has been of inestimable value, but has resulted in disturbing the peace and confidence felt in the accuracy of physical diagnosis prior to the introduction of this new instrument of precision.

The x-ray has taught us much, both on the positive and negative side. It has forced us to unlearn much that we had believed concerning physical examination and physical signs. It has taught us that as a rule we hear much less of lung pathology than really exists. It has taught us that in reading the film we must be careful to sift the grain from the chaff, and that nicety of judgment and experience are requisite in order to obtain an accurate idea of what the x-ray has revealed.

Practically the entire laity and quite too many physicians regard the x-ray as all-revealing, whereas, as a matter of fact, it shows but one thing: differences in density. It remains for man's brain to interpret these differences in density in the light of his knowledge of pathology and of all other evidence to be derived from examination of the patient before alloting to them their true value and worth. Brown has two prognostic theses relative to the x-ray that are so true that they must be quoted:

"He usually does best whose serial roentgenograms, other things being equal, show no further change." (Would that our patients could grasp this!); and "Prognosis is more uncertain as long as the roentgeno-

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gram indicates change in the process for worse or even for better."

General Condition of the Patient.—The general condition of the patient represents that combination of symptoms, both subjective and objective, which shows whether the sick man is mastering the disease or being mastered thereby. Brown says again:

"Physical signs" [to which I might add "and x-ray"] "tell by inference what has happened in the lungs, symptoms what is happening."

The general condition is more important in prognosis than the physical signs or the history. Thus, irrespective of minimal physical signs, coupled with a benign x-ray picture, the patient with a daily fever of 102°, with a pulse persistently 100 or over under absolute bed-rest, with relative or complete anorexia, and with steadily progressive loss of weight, is seriously and dangerously ill. "Symptoms tell what is happening." I often tell patients that the important factor in their outlook is not how much involvement they have as how well they are handling it. As an illustration, I bring to mind two men marching side by side; one is carrying a 50-pound pack with ease; his neighbor is staggering under a 35-pound pack. The latter is in a worse condition despite the fact that his load is lighter.

Thus far the problem is relatively simple. We would naturally be led to suppose that in a bad case there would be present abundant physical signs, extensive x-ray evidence of involvement and marked symptoms of systemic intoxication. Unfortunately, this does not always occur. The greatest discrepancies are found between physical signs, x-ray findings and general condition. As a rule in these cases, x-ray involvement will be alarmingly extensive and the physical signs distressingly absent. The general condition may vary from one of almost florid health to one of obviously hopeless illness.

Economic Situation and Intelligence of Patient.—It is a mournful fact that many patients simply cannot afford to get well of tuberculosis. The economic factor in a disease requiring often two, three or four years of total working incapacity in order to achieve apparent arrest, becomes the dominating element. Our state, county and municipal sanatoria are at present quite unable, for financial reasons, to keep the patient until recovery is attained. A year or eighteen months' residence is all that can be allowed. A patient returns home and attempts home treatment, the difficulties and failures of which I have elsewhere pointed out.

The unintelligent, too, form a large number of those for whom recovery is unattainable. I draw a sharp line of distinction between the unintelligent and the uneducated. They are by no means synonymous. The unintelligent never grasp "what it is all about;" the uneducated, even though almost illiterate, will respond readily to instruction and will take the cure faithfully and well. Of course, the wholly illiterate are under an enormous handicap, as they cannot benefit from the mass of educational literature available to the tuberculous.

Have we considered everything that has a bearing on the ultimate outlook of the patient? Hardly! Two most important factors have not been mentioned, namely:

(1) Virulence of infection
(2) Degree of resistance

The interplay between these two antagonistic forces constitutes the battleground upon which the disease is won or lost.

We talk very glibly about infection and resistance, whereas in reality we know too little about them. We know this about tuberculous infection: that it is remarkably uniform in its virulence and that virulence remains at about the same degree in any individual case, though the structure of tubercle and its resistance to stress varies and is constantly changing, and though the number of infecting tubercle bacilli may vary to a limitless extent.

We know, with reference to resistance, that we are dealing with two forces: immunity and allergy. Krause has given such a good definition of these terms that I quote him:

"By 'immunity' is meant that condition of increased specific resistance to implantation and extension of tubercle bacilli; and by 'allergy' is meant that state of tissue hypersensitiveness to tuberculo-protein that comes into existence with the earliest formation of specific anatomical tubercle."

We do not know the nature of this "specific resistance" that is the keynote to immunity; we know something of the mechanism by which it operates and we know, too, that it varies in intensity, developing positive and negative phases. The factors that bring about variation in the degree of specific resistance we, however, do not know, nor have we any adequate means of measuring this resistance. The various laboratory procedures on the blood, sputum and urine that have been advanced as being of prognostic

importance, have either been of no real aid or else have been merely corroborative of facts gathered elsewhere. None of them has been of sufficient worth to warrant the placing of any great confidence in the results.

There is no doubt that mobilization of bacilli is often the cause of a sudden flare-up and of the development of a tuberculous pneumonia or of a general dissemination. There is unfortunately no way in which we can prevent this mobilization, nor is there any method by which we can discover that such mobilization is imminent.

Krause points out that: "The presence or absence of symptoms is to a large extent contingent on focal mechanics." Increasing fibrosis means a gradual release from symptoms, while increasing degeneration and necrosis, "softening," as we term it, mean added severity of symptoms. The patient who has extensive anatomical tubercle with comparative freedom from symptoms has his tuberculous process well sclerosed and hemmed in. The patient who has marked constitutional disturbance with but slight anatomic change has very slight or imperfect focal investment.

With regard to allergy, we know that it cannot exist in the absence of tubercle. The allergic state is not stable, but is continually fluctuating, depending upon the extent of fibrosis or the amount of absorption from the tuberculous process.

The acute flare-ups of clinical tuberculosis must denote reinfection because they produce symptoms that are of inflammatory origin and so must arise from immune and, therefore, allergic soil. Hence, then, acute flare-ups must be looked upon as immune phenomena; and, granted that the patient can survive their initial intensity, should be a good omen in ultimate prognosis as bearing testimony of increased allergic reaction and, therefore, of proven immunity. It was in harmony with this theory that early workers with tuberculin sought to obtain marked systemic reactions.

Differences in constitution cause individuals to vary in their anatomical responses to tubercle bacilli which infect them. As Krause says:

"It is stretching probability not at all to suppose that the native capacity of tissue to react fibrously to injury of any and every kind varies with the individual constitution. . . . What is, of course, important in this connection is that individual variations of tissue capacity for fibrosis would surely affect pathogenesis in the individual case."

Here is another all-important factor in prognosis which might be denominated the "fibrogenic aptitude" of the patient. In using this expression, I am boldly paraphrasing a title used by our Chairman, Dr. Houston, in a most charming and original paper which he wrote some two years ago called: "The Spasmogenic Aptitude." The presence or absence of the fibrogenic aptitude is of great moment. How can this quality be detected early, so that it may be of some value in forecasting? The fibrogenic aptitude is intimately related to the degree of activity present. The word "activity" is one that has been a bone of contention for some time. It is difficult to secure some standard definition. It has been suggested that an active case may be defined as one presenting symptoms, these symptoms being due to the presence of tuberculosis. So, over and above the history, the physical examination, the x-ray, the symptomatology, and the economic situation and intelligence of the patient, we must consider the grade of immunity and of allergy, the fibrogenic aptitude and the degree of activity before having at hand all the material on which to base a prognosis and, unfortunately, the last three elements (immunity, allergy and activity) are very difficult to estimate.

We are living in an age of functional medicine. We can fairly well estimate the functional capacity of the heart muscle; we can reasonably tell the degree of failure on the part of the islands of Langerhans in diabetes; we can gauge approximately the working capacity of the kidneys in a case of nephritis; but in the infectious diseases, though we have specific therapy against some of them, we have practically no measure by which we can determine just what the fighting reserves of the patient are. That is why we come up against such antitheses as the extensive x-ray picture and the excellent general condition, or the scant physical signs and indisputable evidence of grave toxemia. That is why we never know when a change is going to take place and when, almost literally, "The gladness of the morning will be turned into the evening of sorrow."

I do not mean to be pessimistic; I am merely looking facts in the face and commenting upon them. We see daily problems demanding solution and patients almost piteously begging for assurance for their future. We should not tell all that we know, but most of all we should not tell them all that we do *not* know. To do that would destroy their faith in the profession that

is honestly seeking to aid them. We know something. All our prognoses are not bad. We estimate conflicting evidences correctly in the majority of cases. All of us, for instance, know that very extensive x-ray involvement, even in the presence of scant physical signs and good general condition, demands a most guarded forecast; but the basic nature of the degree of resistance and of its specificity and non-specificity, its brief and lengthy variations, are as yet unknown. As Krause says:

"But tissue cells are the pieces through which the motive forces work. . . . Still further in the future lies the complete and comprehensible demonstration of the energies, the biochemical and biophysical impulsions and repulsions which set all the cells into play. And beneath all is life—whatever its essence may be."

Until the worker in the laboratory unlocks the door to the safe that conceals this most precious and most sought-after secret of nature, we will be obliged to bow before the majestic arm of the Unknown which stands before our finite minds and says: "They shall not pass."

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DISCUSSION (*Abstract*)

Papers of Drs. Baumgarten and Rusk and Dr. Ringer.

Dr. C. H. Cocke, Asheville, N. C.—The prognosis in tuberculosis depends very much more upon what I denominate as qualitative tuberculosis than upon quantitative disease, or extensive involvement of the lungs. Immunity is still poorly understood in this disease, and why apparently similar cases react dissimilarly and proceed to diverse results is still beyond the knowledge of most of us. We cannot at present separate the humoral from the cellular factors in attempting a qualitative estimate of immunity. About the best we can do is to realize always that immunity in any given tuberculous patient may vary tremendously, is sometimes up and sometimes down, and that it is never an absolute quantity. Hence the need for always being guarded in one's prognosis of a given case.

Dr. Baumgarten's paper presents the interesting thesis of an attempt to relieve pleural effusion by obliteration of the pleural cavity, employing a means which up to the present moment has been used for a directly opposite purpose, namely, to effect a pneumothorax by preventing symphysis of the pleura. The original indications for oleothorax and those stressed recently by Ray Matson before the Southern Tuberculosis Conference in Atlanta about four weeks ago were the following:

(a) To attempt to sterilize a pyopneumothorax.

(b) To get a little more effective pressure than one dares to attempt by the injection of air.

(c) To prevent closing of a pneumothorax by adhesions already present.

(d) To cause an increase of rigidity of the mediastinum.

(e) To close a small bronchial fistula.

You will note that the whole effort is at keeping the pleural leaves apart and exerting what pressure one may deem wise on the underlying lung.

Dr. Baumgarten cites two cases of pleural exudate (or transudate), quite probably of myocardial origin, though I note that a later observation states that one case turned out to be malignancy of the pleura.

I am far from being a physiologist and certainly would not pose before you as a myocardial expert of sufficient information to say that this pleural effusion may or may not be a safety valve for an already overworked heart muscle. The suggestion, however, has occurred to me that this may be the case. I am, therefore, wondering what might happen to this damaged heart should this relief be withdrawn. The further observations of Dr. Baumgarten's work will undoubtedly throw further light upon the advisability of his procedure.

Matson shows that about 50 per cent of his cases of oleothorax react distinctly to the use of oil in the pleura. I think attention should be called to the differences in reaction to the vegetable oils commonly used, such as olive oil and paraffin oil, which has been more or less commonly in use. These dangers, of course, have been pointed out by many others; but some may still not be aware that paraffinomas have been observed as a result of the constant, chronic irritation of paraffin oil. These doubtless are quite distressing to the patients. The reaction to the injection of oil into the pleura may be normal or hyperactive, with a resulting chill, rise of temperature and a tremendous sense of discomfort that causes one to hesitate to repeat the injection. I think a safe guide is to inject originally one or two c. c., preferably of the vegetable oil, to find out what is the individual reaction of the pleura. The procedure is not one to be practised by those who are not well skilled in phthisiotherapy.

Dr. L. J. Moorman, Oklahoma City, Okla.—Regardless of what we may think of the physiologic processes, it seems that Dr. Baumgarten has given symptomatic relief to his patients, and he says he has eliminated the psychological strain occasioned by the fear of frequent punctures, also possibly the danger of infection.

However, I must confess I have wondered about the advisability of closing a pleural sac in a case of myocardial weakness. Like Dr. Cocke, I am not sufficiently familiar with Nature's processes to draw definite conclusions; but I wonder if there might be increased danger of edema of the lungs if we close the safety valve in these cases. It is my opinion that the method should have further study before definite conclusions are reached.

Dr. Ringer has pointed out the fact that the foundation of prognosis in tuberculosis is not built upon a rock. It necessarily stands upon shifting sand and must evolve as the patient is observed. I believe this is a very opportune time to refer to the psychological factors in connection with the prognosis.

After all, prognosis depends not only upon the physi-

cian's ability to appraise the case from a pathological and clinical standpoint, but equally important is his ability to deal with the patient as a composite whole, especially from a psychological standpoint. The prognosis naturally evolves as we see the effects of the psychological storms of the patient. The environment and the patient's reaction to it have much to do with the prognosis. All the environmental factors which tend to disturb peace of mind should have careful consideration.

The financial and the family factors often play a large part in the patient's psychology. The physician's own personality and his attitude toward psychotherapeutics may have much to do with the prognosis.

Dr. Wallace J. Durel, New Orleans, La.—The more I delve into the subject of prognosis, the more attention I pay to the monocytic index, actually, to some extent, trusting the monocytic index against facts clinically established.

In cases which seemed clinically hopeless, but in which the monocyte index was high, we have observed recovery. The monocyte index is vital in prognosis.

Dr. Baumgarten (closing).—The criticism of Dr. Moorman is, of course, perfectly well taken and I have no physiological explanation to offer for the result. As a matter of fact, no pulmonary edema did develop in these cases as a result of the procedure; the patients were, on the other hand, in immediate danger of succumbing to the exhaustion incident to the frequent aspiration of large quantities of pleural fluid. This, as was pointed out in the paper, had failed to yield to indirect therapeutic measures. In the meantime exact knowledge of the physiological and pathological interchange of pleural fluid still remains to be worked out and until this is done we shall be more or less at a loss for a satisfactory explanation.

THE ROLE OF THE ROENTGEN RAY IN DETERMINING THE CAUSE OF GASTROINTESTINAL BLEEDING*

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The roentgenologist is frequently perplexed at being unable to demonstrate intrinsic gastrointestinal lesions in cases giving a history either of gross hemorrhage or in which occult blood is found in the stools. It seems not inappropriate briefly to review various factors entering into this question.

Approximately 70 per cent of the patients whose major complaint is gastrointestinal bleeding show no organic lesion in the alimentary tract.

Gastrointestinal bleeding may be evidenced by the presence of blood in the gastric contents or in the stools, either or both depending upon its amount and the location of the bleeding vessel. The blood may be bright red or dark, depending on its source and whether it is digested or indigested.

There are many substances which, when ingested, closely resemble blood, and care must be exercised to avoid mistaking them for blood. The following substances may produce pseudohematemesis, or simulate blood in the stools: the various preparations of bismuth often combined with soda, the iron compounds, cranberries, red wines, beets, spinach and allied vegetables, carrots, cherries, chocolate, red sausage, and others. The initial step, then, in diagnosing any case of this nature is to rule out pseudohemorrhage.

The ingestion of meats, injuries to the gums, overenthusiastic use of the tooth brush and other traumas to the buccal mucosa are so well known as a cause of occult blood in the stools that they need be only mentioned here as possible sources of bleeding.

The existence of true bleeding having been established, the causes may be grouped in relation to the gastrointestinal tract as follows: (1) extrinsic or secondary and (2) intrinsic or primary.

(1) EXTRINSIC OR SECONDARY GROUP

Secondary hemorrhage from the gastrointestinal tract is usually congestive. The congestion may be active or passive.

The commonest causes of active congestion are: infections which produce congestion and irritants such as various caustics, emetics or irritating purgatives. Various substitutes for portable alcoholic liquors have in recent years markedly increased gastrointestinal hemorrhage.

Chief among the infections which produce congestion and subsequent hemorrhage must be mentioned cholecystitis, appendicitis and that type of infection generically termed gastroenteritis.

Gall-bladder disease produces blood in the vomitus or in the stools in 2 to 4 per cent of cases, and 20 per cent of gall-bladder patients show blood upon gastric analysis.[1] Not uncommonly cholecystic disease produces frank hemorrhage.

Diseases of the appendix produce gross bleeding in from 1 to 2 per cent, while in 24 per cent altered blood is found on gastric analysis.[1]

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*Read in Section on Radiology, Southern Medical Association, Twenty-Fifth Annual Meeting, New Orleans, Louisiana, November 18-20, 1931.

*From Radiological Clinic of Drs. Groover, Christie and Merritt.

The true mechanics of gastrointestinal bleeding resulting from gall-bladder and appendical disease is assumptive. However, localized congestion either from septic thrombi or from fermentation due to faulty digestion offers most tenable explanation. The possibility of exaggerated peristalsis, an accompaniment to the reflex pylorospasm seen associated with these diseases, producing capillary rupture, must be considered. Irrespective of the means of production, bleeding associated with either or both of these diseased conditions, is not an uncommon occurrence and is a highly important clinical entity.

Gastrointestinal hemorrhage associated with a Meckel's diverticulum has been reported and this abnormality must be considered as a possible cause. It has been most frequently noted in children.

The clinical picture of infectious gastroenteritis is so well known that it requires no elaboration in this paper.

Passive congestion which may cause bleeding results from practically all hypertrophic conditions of the liver, spleen and pancreas. This is especially true of malarial infections and of that type of splenomegaly seen in Banti's disease. Rarely, post-peritoneal tumors such as hypernephroma or the lymphomata, by their pressure may produce gastrointestinal congestion and secondary hemorrhage.

Passive congestion in these conditions results from the disturbed blood supply. This vascular alteration varies from diffuse hypostasis to the formation of true varices. These are commonest in either the lower esophagus or cardiac segment of the stomach. Not uncommonly following splenectomy there is sufficient disturbance in the blood supply to produce bleeding in the stomach.

There are generalized pathological conditions in which hemorrhage occurs from the gastrointestinal as well as from other mucous membranes, for example: sepsis, the exanthematas, autotoxic states, cholemia, uremia, blood dyscrasias and diseases such as hemophilia, purpura, scurvy, the leukemias, pernicious anemia and various cardiorenal conditions.

Hemorrhage may be incident to various neuropathies, vicarious menstruation and burns.

(2) INTRINSIC OR SECONDARY GROUP

An intrinsic lesion produces hemorrhage by excavation and erosion of some vessel. The degree of hemorrhage obviously depends on the size of the vessel, but not of the lesion. A lesion almost microscopic in size may produce fatal hemorrhage while an extensive lesion may produce only a seepage.

The commonest intrinsic causes of gastrointestinal hemorrhage are: (a) ulcerations, (b) malignancies, (c) benign tumors, and (d) hemorrhoids.

(a) *Ulcerations.*—Ulcers may occur anywhere throughout the alimentary tract, although the commonest site is the first part of the duodenum. Practically all ulcers of the stomach are situated in the vicinity of the lesser curvature and 90 per cent are in the vertical portion of the stomach, that is, that part lying above the incisura angularis. When an ulcer is situated near the greater curvature it is usually malignant.

Not infrequently, multiple gastric ulcers are found, and the association of gastric and duodenal ulcers is well known to roentgenologists. Balfour[2] states that 25 per cent of gastric ulcers produce bleeding. However, irrespective of their location, size or number, the incidence of bleeding depends upon whether or not a blood vessel is involved in the destructive process. This point cannot be too strongly emphasized, especially in those cases where surgical intervention is contemplated.

The site of predilection for ulcers in the gastrointestinal tract is the duodenum. Reeves[7] states that 98 per cent of duodenal ulcers are found within the first inch and a half of the duodenum. The blood supply in this region and for the lesser curvature of the stomach is similar. This may explain the occurrence of ulceration in these regions.

The ulceration of the duodenum may be on the anterior or posterior wall and quite frequently both surfaces are involved. The first part of the duodenum lends itself most easily to both roentgenologic and surgical exploration. Nevertheless, it must be borne in mind that duodenal ulceration may occur deep in the duodenum, even as far out as the ampulla of Vater, and in this location its detection is extremely difficult both roentgenologically and surgically. In fact, unless the process be sufficiently extensive to produce a crater or obstruction its presence will probably not be suspected. These deep ulcerations are especially prone to produce hemorrhage, which is often so great as to be exceedingly alarming.

Twenty per cent of duodenal ulcers produce bleeding. Repeated hemorrhage in the presence of a preceding history of epigastric pain, signi-

fies an ulcer of the stomach or duodenum in 90 per cent of cases.

The small bowel, distal to the first part of duodenum, is comparatively free from ulceration except in typhoid fever and tuberculosis. Tuberculous ulcers rarely produce hemorrhage.

The colon is quite a frequent site of ulceration and ulceration of the colon constitutes the only true form of organic colitis. The ulceration may be tuberculous, amebic or due to various specific organisms such as the Shiga bacillus or various strains of the streptococcus. As in other locations, if a blood vessel is involved, hemorrhage results. The bleeding from colonic ulceration, however, is usually associated with diarrhea and the presence of mucus in the stools. The blood is confined entirely to the fecal excretion.

The majority of the ulcerative lesions of the large bowel give typical roentgen findings and the diagnosis can usually be established by roentgen examination and prostoscopy.

One of the commonest abnormalities found in the large bowel, diverticula, produces hemorrhage only when infected and secondarily ulcerated.

(b) *Malignancies.*—Malignant tumors may occur in any part of the gastrointestinal tract. However, their points of election are the lower third of the esophagus, the stomach and the large bowel, especially its terminal third. Rarely does malignancy primarily involve the small intestine. As a symptom of gastrointestinal carcinoma, hemorrhage must be regarded as a late sign since the hemorrhage is usually the result of secondary ulceration in the primary growth. When it occurs it is usually a seepage as evidenced most commonly by the coffee ground vomitus seen in gastric carcinoma and the blood streaked stools seen in large bowel malignancies. Not infrequently, gross evidence of bleeding is not noted, the loss of blood being evidenced by secondary anemia.

No matter where the location of the malignancy may be, its roentgenologic manifestations depend on a combination of three signs: (1) a filling defect, (2) obstruction to passage of the opaque column, and (3) a loss of mural flexibility. The predominance of any one of these signs over the other depends on the degree of luminal involvement.

(c) *Benign Tumors.*—Benign tumors may occur in any part of the gastrointestinal tract. They are usually single and pathologically they are usually one of the following types: fibroadenomatous polyps, polyposis, adenoma, fibroma, myoma, adenomyoma, fibromyoma, myofibroma, hemangioma, hypertrophied mucosa, papilloma, dermoid cyst, or an hypertrophied pyloric muscle.[4]

When multiple tumors are found they are usually polyps. It must be borne in mind that there is both a pathological and clinical difference between multiple polyps and gastric polyposis, the latter being an extremely rare condition.

Benign tumors are very uncommon in the esophagus and when present in this location they rarely produce bleeding. The symptoms are usually those of obstruction.

By far the commonest site of benign tumors of the gastrointestinal tract is the stomach. However, they are not common in this location. This is evidenced by Eusterman's[4] statement that less than 0.5 per cent of gastric tumors are benign when diagnosed. In spite of their relative rarety, benign tumors of the stomach represent a definite entity and occasion a bizarre chain of symptoms, the most important of which is due to the loss of blood which they produce.

Benign gastric neoplasms bleed by secondary ulceration in the tumor. The bleeding is rarely profuse, is often unsuspected by the patient and is manifested by a secondary anemia so profound that it often simulates the pernicious type. Many cases of benign gastric tumor have been diagnosed and treated for pernicious anemia.

Clinically the diagnosis of benign gastric tumor is most difficult, usually impossible. Roentgenologically, they give a typical picture and the diagnosis can definitely be established in a high percentage of cases.

It may not be inadvisable briefly to mention their roentgenologic characteristics. They are prone to be situated on the walls rather than the gastric curvatures. They protrude into the lumen and produce a clearly defined filling defect. This luminal protrusion causes a sudden separation of the barium column when it reaches them. The walls of the stomach surrounding the tumor show a normal flexibility and the tumor interferes with peristalsis only in its immediate area. Very rarely does it give a palpable mass and when a mass is felt the tumor has probably undergone malignant changes.

Benign tumors of the small bowel are extremely rare. Polyps are found in the first part of the duodenum, where they produce the typical clear-cut filling defect in the duodenal cap. However, most of these are attached by a pedicle to the gastric mucosa and have prolapsed through the pylorus into the duodenum.

The colon is not an infrequent site of benign tumors, usually polyps. These are frequently multiple and give a characteristic roentgenological picture. Apparently in the large bowel they rarely produce hemorrhage and are important chiefly on account of possible malignant changes. The polypoid excrescences of granulation tissue produced by ulcerative colitis must not be confused with trye polyps of the large bowel.

The commonest causes of rectal bleeding are hemorrhoids and anal fissures, and these should always be excluded.

In conclusion, it must be stressed that gastrointestinal bleeding is a symptom and that all diagnostic aids should be employed in the detection of its source. One of the most important of these agents is the roentgen examination.

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DISCUSSION (*Abstract*)

Dr. William Gerry Morgan, Washington, D. C.—All the papers of this program are dealing with subjects which ought to have an appeal for the internist and for the surgeon as well. Dr. McPeak's paper deals with a condition which every internist and many surgeons must see constantly. It is one which often presents great difficulties of diagnosis. It is a condition where the history which the patient gives is of prime importance. It is a condition where we are too prone to jump to conclusions at the outset. It is necessary to rule out all of the conditions that might give rise to hemorrhage from parts of the body that are easily explored with the eye, as Dr. McPeak has suggested, the oral cavity and the anus and rectum and vagina. The proper next step is to turn at once to radiology. Radiology often is able definitely either to determine at once the source of bleeding, or to rule out those more obvious causes for bleeding. I find that radiologists differ in the facility with which they pick up defects which might give rise to bleeding.

I believe that the early ulcer, or fresh ulcer in the stomach, is more apt to give severe hemorrhage. And it is often difficult to discover, even at necropsy. In one case of hemorrhage the stomach was taken out at necropsy and it was reported that no ulcer could be seen. We tied up both ends of this stomach, forced air under high pressure into it, and found on the outside just a little ballooning at a point on the lesser curvature. We opened it and observed a serpentine ulcer with an opening into the stomach hardly larger than the head of a pin. We dissected it out and found a long ulcerated area beneath the mucosa.

Such cases, of course, could be multiplied many times by the surgeons and roentgenologists present. So we see that these cases often do present great difficulties in making a diagnosis. I repeat, one of our great troubles is our tendency to jump to conclusions as to the underlying cause for the hemorrhage, which may come from anywhere in the alimentary tract from the gums to the anus.

Dr. Urban Maes, New Orleans, La.—A general surgeon naturally feels some hesitancy in entering upon the discussion of a subject, even in his own field, which has been as thoroughly covered as this one has been. Dr. McPeak has further proved a point I have made many times before, that the radiologist gets himself into trouble by the very excellence of his interpretation of the findings in disease of the gastrointestinal tract, in that he makes the surgeon lean too heavily upon him. There are few surgeons today, even when they are quite confident of their diagnosis, who are willing to open the abdomen without securing the opinion of the radiologist to confirm their own.

Dr. McPeak, however, in his opening remarks pointed out that at least 70 per cent of the hemorrhages which are the result of disease of the gastrointestinal tract cannot be defined by the x-ray. The radiologist is an expert consultant in his special field, his opinion is of the utmost value, but he should not be asked to achieve the impossible, and he should be given a reasonable amount of cooperation. Not the least of his duties is to confirm or negative the opinion which the surgeon has advanced, and which he has a right to know. His negative report is quite as valuable as his positive report.

He can help us, for one thing, in determining the cause of those diseases which are associated with deformity somewhere in the gastrointestinal tract. He is able to confirm or deny the existence of ulcers of the stomach or duodenum, for instance, or carcinoma, particularly carcinoma of the large bowel, which may give rise to hemorrhage in the early stages before it gives rise to any other symptom.

We are many times justified in resorting to exploratory operation, even in the face of negative radiologic findings, if our experience and our judgment persuade us that this is the proper cause. Instance after instance might be quoted.

There are also cases of intrinsic gastric hemorrhage, bleeding whose origin cannot be traced. Eight cases of this sort have been reported by Archibald. These are the patients, of course, who need transfusion and not surgery, but to make the diagnosis before operation is almost impossible.

Carcinoma of the large bowel is difficult to diagnose radiologically in the early stages if it is of the intrinsic variety until it produces enough deformity by infiltration of the walls or constriction of the intestinal lumen to proclaim its presence. We cannot expect the radiologist to diagnose such cases for us.

When we call on the radiologist for his opinion, we ought to remember the 70 per cent of the cases of gastrointestinal hemorrhage which, for all his skill and

experience, he cannot diagnose for us. The x-ray has by no means supplanted the history and the physical examination in the making of a diagnosis. In diagnosing gastric ulcer, for instance, the history comes first, but the x-ray comes next, and neither is complete without the other. The surgeon and the radiologist are interdependent, and only by cooperation betwen them will the patient's best interests be served.

THE MANAGEMENT OF TOXIC GOITERS*

By ADDISON G. BRENIZER, M.D.,
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This series of 2,500 thyroidectomies includes unilateral and bilateral lobectomies in 1,482 cases of exophthalmic goiter, and enucleations, unilateral and bilateral resections and dissections in 1,018 adenomata, cysts, and cases of simple goiter and thyroiditis.

Females predominated in the proportion of 2,238 to 262 males.

Deaths occurred in 12 cases, 10 of exophthalmic goiter and 2 of adenoma. There were 3 late deaths in cases of recurrent carcinoma, which has made up about 1 per cent of the group of 1,018 cases.

There were 47 cases operated upon elsewhere in the whole series of 2,500, requiring a second operation by us. Of the 2,500 cases operated upon by us, 13 cases, 9 of exophthalmic goiter and 3 of adenoma, have required re-operation, and 2 of these were re-operated upon twice on account of hyperthyroidism. The metabolic rate in all of these cases was above plus 40.

We have found but one case of permanent hypothyroidism following all operations for exophthalmic goiter and toxic adenoma, and certainly no case of myxedema. There have been, however, a number of cases of transitory hypothyroidism which have finally been stabilized by withdrawal of postoperative iodine administration or the taking of thyroxin over a short period of time. There has been one death from severe and persistent hypoparathyroidism and tetany, and three patients showed mild transitory symptoms of hypoparathyroidism. There is no case of permanent aphonia. There are, however, 4 cases with altered voices and lameness or paralysis of one vocal cord.

In the 13 cases where second or third operations were done and in the 2 operated upon twice, making 17 second or third operations on 13 cases, all but one have returned to a satisfactory state of thyroid equilibrium, judged by clinical symptoms and metabolic rate. There is no evidence of hypothyroidism, nor hypoparathyroidism, nor alteration of voice after these 17 secondary thyroidectomies. The one case, after three thyroidectomies, is still mildly hyperthyroid with a metabolic rate of plus 24, but his clinical improvement is altogether satisfactory.

Our operation in cases of exophthalmic goiter for the last fifteen years has been almost invariably as follows: The usual collar incision is made, with dissection of flaps between the platysma and underlying muscles, incision up and down between the muscles, rarely with transverse cutting, and the gland is exposed. The isthmus is bluntly elevated from the trachea, and when possible caught with two clamps and cut. Immediately one begins to undercut the two lobes, leaving a thin wedge of gland on either side of the trachea. Then working outward and from the lower pole upward, each lobe is raised from its capsule, the superior vessels are easily recognized, and by still undercutting and elevating, all the gland is removed except the wedge-shaped strips along the trachea and a piece of the upper pole, the size of the first joint of the little finger.

At the second or third operation, the wedge-shaped strip and portion of the upper pole have been found much enlarged on one or the other or on both sides, as large as the gland at many primary operations. After separation of the sterno-thyroid muscles from the front of the trachea and gland, mostly by sharp dissection, the lobes are again undercut from the midline outward and elevated from the lower pole upward and outward to the upper pole, with difficulty on account of adhesions, but in the same way as at the primary operation. We have but one case of postoperative hyperthyroidism and no other particular disturbance in the postoperative course of these 13 cases which have undergone more than one operation, in spite of the fact that metabolic rates ranged between plus 42 and plus 65.

At all operations for adenoma we have inspected, palpated and even incised every portion of the gland left behind for additional smaller adenomata, and in spite of this diligent search have had three cases of growth of an adenoma

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evidently left behind. One of these we operated upon three times, at each time removing additional adenomata. This was not carcinoma, as we first feared. It was proved not to be by pathologic section, confirmed by the fact that the man is living and in good health eight years since the last operation. These cases of adenoma were not re-operated upon because of recurrent masses in the neck, but on account of elevation of the metabolic rate above plus 30.

It is made clear from the above report and description of operation that the number of cases of both recurrent hyperthyroidism and hypothyroidism have been comparatively few; that the chances of hyperthyroidism are about four to one over hypothyroidism, in spite of the fact that the operation is radical, leaving a minimum quantity of the gland behind. This procedure at first, second and third operation has been safe and has given almost absolute protection to the recurrent laryngeal nerves and parathyroids.

Whatever the exact amount of gland left behind in resections of the thyroid, it should be the minimum for the sure relief of exophthalmic goiter, but also an amount sufficient to cover and protect the recurrent laryngeal nerves and parathyroids; and with these far more important factors kept firmly in mind, the question of hypothyroidism is not to be considered at the time of the operation.

The occurrence of goiter in our series is about 10 to 1 in the woman. In another paper I have drawn the following conclusions: (1) Cystic, fibrocystic ovaries and ovarian cysts are not infrequently found in association with hyperthyroidism. (2) At puberty, menstruation, the menopause and during pregnancy, thyroid activity is inversely proportional to that of the corpus luteum. When the corpus luteum is adequate, there is little or no enlargement of the thyroid.

During the war we observed a sequence of effort syndrome, shell shock and exophthalmic goiter, and 14 of these cases demanded operation, although our attitude was not to operate upon them during the period of war activities, but to retire them as unfit for further service.

The use of iodine in adolescent goiter, in exophthalmic goiter, in toxic adenoma and as a postoperative protection against regeneration of thyroid tissue and recurrence of hyperthyroidism, is too well known for remarks at this point beyond the comment that iodine is also a two-edged blade, frequently arousing non-toxic adenoma to activity and when prolonged bringing about exophthalmic goiter, as we have observed several times in simple adolescent goiter.

The question of infection in exophthalmic goiter has been dealt with by various authors, and toxic thyroiditis has been discussed. I have reviewed the subject of thyroiditis accompanied by hyperthyroidism, with these conclusions: thyroiditis probably makes up 0.5 to 1 per cent of all operative material and 0.25 to 0.5 per cent of all observed clinical cases. The two most frequently proven types of inflammation are tuberculous and woody thyroiditis. Syphilitic thyroiditis certainly occurs. Non-specific thyroiditis has shown its bacterial cause in some suppurating cases. Hyperthyroidism may accompany any type of thyroiditis at any stage, usually the subacute. The relation in tuberculous thyroiditis is striking. Most cases of non-specific and woody thyroiditis finally become hypothyroid, whether operated upon or not. Cases of tuberculous thyroiditis operated upon have given the best functional lasting results. Syphilitic thyroiditis has been relieved with appropriate treatment. Therefore, the more slowly progressive and destructive the type of inflammation is, the more apt it is to be accompanied pathologically by hyperplasia and symptoms of exophthalmic goiter and to give better functional results after operation. The usual amount of thyroid should probably not be removed, even in tuberculous thyroiditis, for at least one case out of 13 has resulted in myxedema.

Crile[4] finally resolves exophthalmic goiter into a dual disease of hyperthyroidism and hyperadrenalism, both factors playing necessary roles. The only factors known to him clinically that can precipitate a thyroid crisis are: (a) pain, (b) emotional excitement, (c) foreign proteins, autointoxication, wound secretion, focal infections, infectious diseases, (d) asphyxia, (e) inhalation, anesthesia, (f) hemorrhage, and (g) the injection of adrenalin. The common factor in these conditions is adrenalin, and, moreover, no other recognized clinical condition causes an increased output of adrenalin.

The same factors, namely, a woman, and usually a woman with a hypo-ovarianism, a shortage in iodine, a focus of infection and a so-called thymo-lymphatic tendency, contribute to the intense stimulation of thyroid tissue, altering the tissue itself so that hyperplasia and hypertrophy of the cells, with decrease of the normal colloid and an oversecretion and, usually, altered

secretion of these cells, still prevail whether the thyroid gland has been reduced in quantity or not. Pemberton[5] cites 3 cases of hypothyroidism and myxedema, following subtotal thyroidectomy for exophthalmic goiter, where this condition recurred, and in one case a second operation was done.

Crile[4 6] experiments and speculates interestingly:

"The thyroid is governed, at least largely, by its innervation; innervation is regulated largely by the adrenals; the adrenals are controlled by the brain and nerve centers, though chemical activity probably plays a role. The thyroid certainly cannot originate hyperplasia, the thyroid has hyperplasia imposed on it; the adrenal glands cannot stimulate themselves, stimulation is imposed upon them; the nerve receptors cannot engage in self-excitation, excitation is imposed on them by physical and chemical forces of the external and internal environment. The entire nerve mechanism, the adrenals, the thyroid, and in a secondary sense other ductless glands, act reciprocally; that is, nerve tissue, the adrenals and the thyroid tissue collaborate in a stepping-up process, a stepping up of activity. In certain individuals, because of some previously existing unknown factor (thymo-lymphatic tendency, or more loosely thyroid-constitution), some chemical or physical unbalance, the normal controls become inefficient or are lost, and a reciprocal excitation continues after the environmental excitation that initiated the activity has ended."

"Peptic ulcer can be controlled by excision of sufficient of the thyroid gland to produce hypothyroidism and administration of enough thyroid extract to permit normal function."

"In 5 cases of intractable or recurrent ulcer of the stomach, unilateral adrenalectomy and partial thyroidectomy decreased gastric and intestinal motility and were followed by disappearance of symptoms."

A patient, however depleted and emaciated, who does not show a marked increase of systolic pressure and pulse pressure, is a warning to us in two ways: first, that the fast heart, tremor and weakness are not due to hyperthyroidism and hyperadrenalism; and, second, if there is considerable increase in the metabolic rate, that this case is terminal or of the apathetic type described by Lahey, and may offer surprising reactions. We fear most the gastrointestinal type of exophthalmic goiter with vomiting and diarrhea, even more than the extremely excited type and the cardiovascular type with fibrillation, for we have in rest, bromides, iodine, digitalis and quinidine and fluids more efficient means of combating the two latter types.

Everybody dealing with the goiter question to any extent has observed the striking clinical benefits and reduction of the metabolic rate in exophthalmic goiter and in certain toxic adenomata from the use of iodine, since Plummer in March, 1922, began its use at the Mayo Clinic.

Rienhoff[7] has shown the tendency of reversion of the pathological picture of the exophthalmic goiter type to the normal or colloid type under the use of iodine. Why the improvement is so temporary and why the action of iodine is so difficult the second and third time after the initial dosage, and is so variable in adenomata, has not been satisfactorily explained. Whether iodine is acting on an excess of normal secretion or an incomplete or altered product of secretion remains to be determined. It has been shown that the toxic gland contains only from one-tenth to one-half the amount of iodine in the normal thyroid gland, and perhaps the supplying of iodine might put the gland at rest from its over-stimulated condition in the effort to produce its incomplete secretion. On the other hand, iodine will not infrequently arouse non-toxic adenomata to activity, will fail to benefit toxic adenomata, at times arousing it to further activity, will most regularly reduce goiters of adolescence; will reduce slowly or not at all simple colloidal goiters of maturity; will arouse certain of these to overactivity, and if given constantly and in overdosage can convert even the simple watery colloidal goiter of adolescence into the exophthalmic goiter type. We have seen such an occurrence in several young girls. We have broken the resistance to iodine in several cases of fulminating exophthalmic goiter and in violent postoperative hyperthyroidism by the giving of as much as 100 drops of Lugol's solution intravenously.

We give iodine in small doses, covering the menstrual periods, for 10 or 12 days out of the month in simple goiter of adolescence and maturity; we do not give iodine in non-toxic adenomata; we do give iodine rarely and then with caution and under observation in toxic adenomata; finally we always give iodine in exophthalmic goiter, but with the warning that it be not administered from the grocery store, and that it be not taken at all unless on the way to operation.

We adhere to the opinion that adenomata represent a response of fetal or interstitial cells to a given stimulus, while exophthalmic or colloid goiter represents the response of the adult cells to such a stimulus, and while these fetal and adult cells become histologically the same in appearance and react in a similar manner, they represent two different processes of development, rather than two stages in the same process. That

the cells of fetal or interstitial origin are more labile than those of adult cell origin is also suggested by the greater tendency to malignancy in adenomata.

Wilson[8] found the incidence of malignancy as follows:

"In Berne, one case of malignant tumor of the thyroid was found in every 93 post mortems; in Prague, one in every 452 cases; in San Francisco, one in every 211; in the Mayo Clinic, one in every 292."

Graham[9] states that at least 90 per cent of the malignant tumors arising from the thyroid epithelium have their origin in pre-existing adenomata. Balfour[10] says that the treatment of cancer of the thyroid should be that of the treatment of precancerous lesions elsewhere, namely: prompt surgical removal of the precancerous condition in the thyroid, the adenoma. Since clinically we cannot always diagnose malignancy in adenomata, and since it is even difficult to say from microscopic study whether an adenoma is malignant or not and we may have to judge by the invasion of the blood vessel walls alone, the treatment of all adenomata, toxic or non-toxic, except in the very young, resolves itself into their surgical removal and, at the same time, a careful search over the apparently uninvolved portion of the gland for other smaller nodules.

There are mixed types of combinations of simple and colloid goiters with adenoma in about 20 per cent of cases, adenomata which appear even in adolescent goiters, as the surrounding enlargement subsides and actually adenomata in the hyperplastic and hypertrophic type of exophthalmic goiter. Actually the differentiation of toxic adenoma from exophthalmic goiter may present difficulties: usually the palpable nodules of the adenoma will distinguish the two, but if these nodules are deep in the thyroid of exophthalmic goiter or ectopic in position, retroclavicular, retro-sternal or intra-thoracic, or if the thyroid of exophthalmic goiter is not hard, finely granular and smooth and uniform, unanticipated difficulty may be encountered.

The difference in the symptoms of thyrotoxicosis in the two conditions is one of duration and degree. In the adenoma it is of less severity and more prolonged, beginning usually later in life. We have found, however, many exceptions to the occurrence of toxic adenomata "later in life" and believe the lateness in life has been over-stressed. We believe that the giving of iodine postoperatively is of little or no benefit in preventing thyroid regeneration and recurrence of exophthalmic goiter; we believe that the recurrence takes place in spite of the iodine and are not so sure that it is not occasionally, in part, responsible for the recurrence. Certainly iodine is of only temporary benefit in the recurrent cases and never so effective as before the first operation. Furthermore, we believe iodine interferes, at least to some extent, in the adjustment of the temporarily hypothyroid cases to a state of approximate stabilization and normality.

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DISCUSSION (*Abstract*)

Dr. Charles E. Waits, Atlanta, Ga.—The patient with extreme hyperthyroidism of rather long duration usually presents a history of having been treated medically over a period of months or years, probably over-saturated with iodine. She is often in a state of poor nutrition and her basal metabolic rate has become more or less fixed at a relatively low level. This fact, together with the patient's pulse rate and seemingly good composure, may easily deceive one as to the ability to undergo thyroidectomy. It is this type of patient who, in spite of careful preoperative preparation and postoperative management, may develop an acute postoperative exacerbation of hyperthyroidism which occasionally terminates fatally.

There is an extreme type of hyperthyroidism associated with adenomatous goiter, myocardial weakness, auricular fibrillation and toxic psychosis. These patients should receive not only the most careful attention of the surgeon, but also the counsel of a competent internist in determining their general physical resources.

The third type of toxic goiter which sometimes taxes our surgical judgment is the patient who develops some other acute surgical condition which demands immediate operation. The hyperthyroidism may or may not have been suspected. If it is overlooked one may lose occasionally a patient from acute exacerbation of hyperthyroidism who might have been saved had the condition been recognized and properly managed.

In conclusion, I wish to call attention to the management of the patient who has toxic goiter with a well marked degree of thyroiditis. I think the usual practice of removing a maximum amount of tissue in these patients should be altered to some extent. We ought to leave from one-fourth to one-fifth more tissue to avoid postoperative myxedema.

Dr. Willard Bartlett, St. Louis, Mo.—The disposition of the isthmus at operation should vary with its nature. If one has a very thin layer of tissue over the trachea, the lateral lobes can be nicely isolated and resected separately, leaving the trachea protected. On the other hand, one encounters often a goiter which hugs the trachea like a horseshoe and the isthmus of which is as thick as the lateral lobes. Such a gland I prefer to remove *en bloc*, as does Dr. Brenizer.

In passing, I shall take exception to the essayist's use of the terms "exophthalmic goiter" and "toxic adenoma," not for his benefit, because he is using the terms only for convenience of expression, but because those of you whose interest is primarily in other fields may be unfamiliar with the views on pathology of Allen Graham, Hertzler, and Rienhoff, all of whom have offered convincing evidence that the fundamental pathologic picture lies in hyperplasia of the gland epithelium whether it occurs within gross nodules or in a smoothly enlarged thyroid.

Let me touch on the use of iodine. We use Lugol's solution, of course, as Dr. Plummer advocated. The trouble is that all the other physicians who have ever seen that patient have given it to her and, if they have not, the patient has been buying it at the drug store for a year because she read in the newspaper that "iodine was good for goiter." Consequently, the patient comes to us iodine-fast and is worse off than if she had never had it, because we cannot repeat the iodine effect. We had occasion to study all goiter deaths occurring in our practice between 1925 and 1930. Of these, 38 per cent died before any operation was performed. All had had iodine either before coming to us or while under our care. Iodine worked no miracles there! The answer is, of course, that iodine is badly abused. It should be given only when the patient is being prepared for operation, whether in the home or in the hospital, and should be given after operation to prevent compensatory hyperplasia of the residual thyroid tissue, as advocated by Dr. Else, of Oregon.

The last point is recurrence. I will leave out of consideration those who have had resection of an inadequate amount of tissue except to say that the recurrences from other clinics we encounter are almost invariably in upper poles which, we find at reoperation, have obviously never been touched before. In our opinion, one must deliver and remove the entire upper pole to avoid leaving hyperplastic tissue above an intact blood supply. Dr. Brenizer advocates removing more thyroid tissue than does Pemberton, and I believe that he is more nearly right. At any rate, the crux of the true recurrence is the fact that the individual who develops a toxic goiter, in our experience, has borne that susceptibility from birth and continues to bear it after operation. Removing the goiter will not make her a different individual. If she goes back after operation to the same strain under which she once before developed toxic goiter, whether it be emotional stress, work beyond her nervous and physical resources, or intercurrent infections, she runs a chance of becoming thyrotoxic again. With this in view, we tell the patient: "Getting you back to 'as good as ever' will take about a year. That is true in all severe diseases, whether they require an operation incidental to treatment or not. At some time during that year you will have a mechanical job done that takes only a few minutes, but it will only be an interruption of your rehabilitation and whether that operation is done in a week from now or a month or in three months we shall have to keep you under supervision at intervals until the end of our year. (We used to call that after-treatment.) Then you will enter the maintenance period and it will be up to you; after all, you have learned in this year to stay well. You can do that if you will stay within your own limits, not within some one else's limits." Of course, the majority will get well after operation, anyway, but the particular patients with whom we have to deal illustrate strikingly the fundamental soundness of this point of view.

Dr. Brenizer (closing).—Never have I believed the ordinary statement that the cause of exophthalmos is the contraction of a paltry, little old muscle of Mueller or Landstrom. I do not think the muscle could do it.

What is the cause of exophthalmos? Nafziger, in San Francisco, had a persistent case of marked exophthalmos. I had one, not only like the edema case, but this woman's eyes extended beyond her lids. She burst one eyeball and probably if she had not died she would have done the same thing to the other.

What is the cause of exophthalmos? It is very probably an edematous condition. Nafziger went into the orbit, did a decompression of the orbit and found everything edematous in there. The exophthalmos retired after decompression and drainage. The woman was well satisfied and came back to have the operation done on the other side. Exophthalmos is an edematous condition pushing the eyeball forward. It is very likely that when the outgo of blood to the orbit is much more rapid than the intake, you get a condition not different from the pulsating exophthalmos.

I do not think any of you will be willing to swallow the statement that exophthalmos is caused by contraction of a muscle. I have never seen the muscle. Exophthalmos is an edematous condition.

THE END RESULTS OF TREATMENT FOR PEPTIC ULCER*

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For prognosis in peptic ulcer, statistics up to within the past few years do not prove wholly adequate, for the reason that preoperative and postoperative treatment as well as medical management of peptic ulcer have undergone great changes and vast improvement in the past decade. Hence, in discussing the results of all types of management of peptic ulcer this fact must be kept clearly in mind. Arguing from this basis, an enthusiast might easily be led to predict a greater measure of success for the treatment of peptic ulcer in the immediate future than clinical results might ultimately justify.

Until comparatively recent times, in most instances, patients suffering from digestive disorders were given no comprehensive study before a method of treatment, medical or surgical, was instituted. It was considered sufficient to have made a diagnosis of peptic ulcer. Frequently no differential diagnosis was made between duodenal and gastric ulcer. Often little thought was given as to whether other disease entities entered into the picture, nor was it thought necessary to determine the exact location and condition of the ulcer. The laboratory investigations in most instances were limited to an examination of the gastric contents, the blood, urine and feces and sometimes gastrointestinal x-ray study; and even in the case of the roentgen ray study the giving of antispasmodics to differentiate deformity due to spasm from that due to organic lesion was rarely carried out. No study was made of the emunctories to determine their physiological condition; nor investigation as to toxemia arising from stagnation in any part of the alimentary tract, or to determine the presence of focal infections. All these investigations and more are now carried out as routine measures in the study of patients suffering from digestive disorders before the type of treatment is decided upon.

Such preparatory investigations tend immeasurably to promote the success of whatever treatment is indicated, and so to bring about a larger percentage of successful end results. In cases where medical treatment is indicated such studies enable the clinician to watch the progress of the cure more intelligently. Where surgical intervention is to be undertaken careful preoperative investigation is of even greater importance, since it oftimes corrects functional disorders, and the risk of operation is materially reduced. The exact location and extent of the lesion can now be determined with great accuracy by expert roentgenologists in approximately 96 per cent of cases. With this information the surgeon is often enabled to decide in advance the type, kind and extent of the operation which he will undertake in a given case.

The consensus of opinion is that all uncomplicated duodenal ulcers, unless of the recurrent or hemorrhagic type, should receive medical treatment. Since attempts at healing normally take place in any ulcer, it follows that many ulcers will heal without any treatment at all. Therefore, it is agreed that when an ulcer is encountered in its early course, every opportunity should be afforded for spontaneous healing. A dietary regimen alone may enable an ulcer to heal in a short period of time without recurrence of symptoms. Such treatment should be continued for several months after all symptoms have disappeared.

Although gastric ulcers may and often do heal spontaneously with or without treatment, nevertheless from my own experience I am inclined to view them always as surgical cases from the outset. I take this view because a certain proportion of ulcers of the stomach which in the beginning are diagnosed as benign later prove to be carcinomatous. As yet we have no means of positively differentiating, in the early stages, the benign and the malignant type of ulceration of the stomach, and since only in the very earliest stages of gastric carcinoma is a permanent cure possible, it is important that surgical intervention should be instituted.

Surgical intervention is indicated in the hemorrhagic type of peptic ulcer, but never during the period of bleeding. In the recurrent type of chronic duodenal ulcers and in the presence of permanent stenosis of the pylorus, surgery should be resorted to; the same is true of penetrating and perforating peptic ulcers. Ulcers occurring at the site of the gastroenterostomy stoma should, in a large percentage of cases, receive surgical treatment.

Inasmuch as etiological factors have some

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bearing upon the ultimate success or failure of treatment of peptic ulcer, it may be of interest to consider briefly this phase of our subject. It is not easy to determine the cause in relation to the incidence of peptic ulcer, because often it is impossible to learn even approximately just how long symptoms of ulcer have existed. We may get a history of long duration of digestive symptoms. The most we can do in regard to age is to accept the age which the patient himself fixes as the time of onset of symptoms. Comparatively few cases have been recorded in individuals under ten years of age. The largest number occurs between the ages of twenty and fifty.

The consensus of opinion is that occupation plays a very small role in the development of peptic ulcer.

The excessive use of alcohol and of tobacco, while they probably are not etiological factors, nevertheless act quite definitely to prevent healing.

It is my impression that heredity plays a part in the etiology of peptic ulcer only in so far as environmental influences and hereditary habits may to that extent contribute to the development of ulceration. Environmental influences are of some definite significance in predisposing individuals to disorders of the alimentary tract. During the early years, habits of life, including diet and hygiene, are formed which are continued into adult life and which may, and probably do in many cases, play an important part in the causation of peptic ulcer, as well as of other disorders.

Diet undoubtedly plays an important role in the causation of ulcer in two ways: first, by lowering of body resistance where the diet is deficient in certain necessary elements, and secondly, by injury to the gastric and duodenal mucosa from the ingestion of unsuitable, irritating articles of food.

There can be little doubt that injuries to the mucous membrane of the duodenum and stomach from irritating foods and chemical poisons, among which I would include alcohol and tobacco, predispose to the development of ulcer. On the other hand, external trauma rarely acts as an etiological factor.

For the past fifteen years there has been accumulating gradually more and more evidence as to the etiological role which infections play in the production of peptic ulcer. That any one definite type of infection has a peculiarly selective causative action has not been definitely proven. The source of infections which play an etiological role may come from a focus around the teeth, from the tonsils, from the gall-bladder, from the appendix and even from such remote regions as the pelvic organs in the female and the prostate in the male. It is possible that focal infections in the urinary tract may play the causative role. That such infections as influenza, typhoid fever, malaria, typhus fever and periostitis may play an etiological role in the formation of peptic ulcer, by lowering body resistance, seems within the realm of reasonable possibility.

The immense amount of research work which is being carried on in this particular direction at the present time will in all probability, in the near future, stabilize our views as to the extent of the role which infective organisms play in the etiology of peptic ulcer.

I should hesitate to give anemia any significance in the etiology of gastric and duodenal ulcer, because it is probably, in the great majority of cases, a result of the disorder rather than a precursor of it.

In discussing the end results, or the efficiency of medical treatment of peptic ulcer, the student of statistics considers first the immediate results. What constitutes a cure is still a moot question among authorities writing upon this subject. I believe a patient remaining symptom-free for a period of five years ought to be considered cured. It is exceedingly difficult in many cases to determine definitely whether the recurrence of symptoms is due to a lighting up of the old ulcer, or to some entirely different disease entity. We cannot always be sure that recurrent symptoms may not arise from the development of an entirely new lesion having no relation to the initial ulcer.

Perhaps the most comprehensive study which I have been able to find of the medical phase of the question is that published by Dr. David Smith in the *British Medical Journal* in 1928.

In his review Dr. Smith analyzed 214 cases, 96 male and 118 female, which had been treated from five to fifteen years prior to 1927. With commendable effort he had been able to ascertain the end results over the periods given in all of these cases, so that his conclusions may be looked upon as representing the average end results in cases treated medically. Dr. Smith studied the records in these cases critically from the clinical, radiological and chemical aspects so that the diagnosis of peptic ulcer cannot be doubted. He is careful to point out that he did not attempt to differentiate duodenal and gastric ulcers, although in 602 necropsies 6.8

per cent of duodenal ulcers were found against 5.15 per cent gastric ulcers. This, of course, does not represent the relative incidence of gastric to duodenal ulcers. Most of the cases considered in his series were from among the working and the poorer classes.

Dr. Smith gives as the result of his study of immediate results of medical treatment, that is, at the time the patients left the hospital, the following percentages: males, 67 per cent cured; 22 per cent improved; 3 per cent failures; 6 per cent irregular dismissals and 2 per cent deaths. Of the females, 76 per cent cured; 14 per cent improved; 2.5 per cent failures; 2.5 per cent irregular dismissals and 5 per cent deaths. From this we see that a large percentage of these patients left the hospital either cured or improved. The average period of treatment was forty-three days. Dr. Smith points out that his results correspond closely with the figures given by von Leube in his series of 424 cases. Some later writers show a higher percentage of medical cures, even as high as 90 per cent. In discussing his statistics Dr. Smith draws attention to a fact which coincides with my observation, namely: that better results are obtained from medical treatment in patients whose symptoms have not been present for longer than one year; and he further observes that the reason for a higher percentage of immediate cures in the female is probably the fact that male patients are less patient and seek operative relief much earlier as a rule than do female patients.

Turning to a consideration of David Smith's review of the results of medical treatment at the end of a five to fifteen-year period, we see of the males 29 per cent cured; 15 per cent improved; 31 per cent failures; 6 per cent lost trace of and 19 per cent deaths. Of the females, 40 per cent were cured; 20 per cent improved; 25 per cent failures; none lost trace of and 15 per cent deaths. He classes as failures those patients who subsequently sought surgical relief.

These figures relating to deaths cannot be accepted as representing failures of treatment, because all deaths which occurred among these patients from whatever cause are included. The criteria of cure were the relief of symptoms and the ability to return to their accustomed occupation of whatever nature it may have been. It is of interest and of some importance in evaluating Smith's results to know that in the main his treatment consisted in intensive administration of alkalies together with a bland diet of frequent small meals.

Dr. Frank Smithies, in a paper published in April, 1917, analyzed a series of 470 patients of his own who were treated medically, which gave 77 per cent immediate cures and 23 per cent failures. In another study which he made in 1927 of a large number of cases treated medically he again states that 77 per cent of his cases of this series were cured.

In evaluating the end results of surgical treatment of peptic ulcer, I shall quote briefly from the published reports of Dr. Donald C. Balfour, of the Mayo Clinic, Lord Moynihan, of Leeds, and of Dr. A. J. Walton, of the London Hospital.

In October, 1930, Dr. Balfour reported the results in a series of 500 cases in which relief of symptoms followed operation in 87 per cent. In 69 per cent the relief was either so complete that the patients paid no attention to diet or habits of living, or dyspepsia was so slight as to be controlled by simple measures. In 18 per cent the result could be classified as fair. Of the 69 per cent I would class as only fair the results of all those who had symptoms sufficient to require any measures, however simple, for the relief of dyspepsia. It is only fair to state that other diseases may be responsible for symptoms following gastroenterostomy. As Balfour suggests, "The causes of dyspepsia are so manifold that it cannot be expected that gastroenterostomy would protect the patient against all such possibilities." This also might be applied with equal force to the occurrence of digestive symptoms following medical treatment for peptic ulcer, notwithstanding that a perfect healing of the ulcer is obtained. In this series of 500 cases Balfour finds that 13 per cent of the patients did not obtain permanent relief from the operation. He suggests a number of causes for failure, such as "failure to remove the appendix, functional disorders, and marked indiscretions in the habits of living, particularly the excessive use of tobacco or alcohol, and unwillingness to carry out a reasonable regimen following operation."

In this series of 500 patients, death from all causes was 4.28 per cent, which represents about that of this country at large. The operative mortality in his series was 1.8 per cent. In this series as in all other series studied by him, perforation, either acute or subacute, did not occur in a single case after gastroenterostomy, proving apparently that gastroenterostomy afforded complete protection against perforation. In 9 per cent of his series one or more hemorrhages occurred after operation, with one fatality. He lays some stress on the fact that these hemorrhages followed some unusual physical or mental strain, or overloading the stomach, or the excessive use of tobacco or alcohol. This again illustrates the truth of my assertion that postoperative care of the present day will definitely improve the statistics of the end results of the surgical treatment of peptic ulcer in the future.

Turning for a very brief reference to the end results as viewed by Lord Moynihan, we find in the series of cases reported by him that 90 per cent of "good immediate results" were obtained with 4.5 per cent deaths. Gastroenterostomy was the operation chosen in 95 per cent of the cases. He says that medical treatment was necessary in 4.5 per cent of the cases after operation, and "it should be a matter of routine in all cases."

If and when this dictum of Lord Moynihan is followed universally after operations upon the gastroenteric tract there will be vastly fewer failures following this line of treatment of peptic ulcer. I emphasize this because just recently some authorities are advocating shortening the period and the rigor of routine treatment following such operations.

Now turning to A. J. Walton's studies, we find in a

series of 172 cases studied that good immediate results were obtained in 88 per cent of the cases operated upon. Death as result of the operation occurred in 2.3 per cent of the cases.

Because the question of permanent curability of peptic ulcer presents itself almost daily in my work in the light of fitness for undertaking important assignments to duty, I have given a critical study to the subsequent health histories of such of my patients as I was able to get in touch with who had been under my treatment for peptic ulcer during the period covered from 1904 to 1924. It may be appropriate to state here that all ulcer patients are required to return to the office for observation from time to time and are not discharged until they are symptom free and considered cured. The majority of the cases studied in this series fell into the period between 1905 and 1915.

The following questionnaire was sent out to 500 patients:

Do you consider that you were cured of your ulcer?

Have you had return of symptoms?

If symptoms returned, when?

Have you been treated by any other physician for digestive disorders?

If so, when and for how long a period, and by whom?

What was the nature of that treatment?

Have you suffered from any other ailment?

If so, what was its nature?

What is the condition of your health at the present time?

Date..... Signed.....

One hundred and fifty replies were received at the time of the writing of this paper. From the answers received the fact has been established that all those who considered themselves cured have been physically able to carry on their accustomed routine of life. In other words, the individual who has suffered from peptic ulcer and been cured, finds himself unimpaired for the performance of his life's work. This is an undeniably important fact to establish.

The diagnosis of peptic ulcer was established by critical study of the family history, past history, the history of the symptoms complained of at the time of the examination, analysis of gastric contents after an Ewald test meal, study of the duodenal contents, feces, Einhorn thread test, a routine blood study and a blood Wassermann. Roentgen ray examination of teeth, chest, gastrointestinal tract, including the administration of belladonna for differentiation between a functional and an organic disorder; and finally, a careful physical examination. The patients who comprise this series were individuals from all walks of life, but the majority were Government employes.

The average age of my series of patients, at the time they presented themselves for relief of symptoms, was thirty-one years. No attempt was made to estimate the actual age at the time of onset of symptoms for the reason that that is distinctly a relative matter. Thirty-four occurred between the ages of twenty and thirty, and 44 between the ages of thirty and forty. In other words, one-half of the series fell into the third and fourth decades of life; the youngest was eleven and the oldest seventy, as shown on *Table 1.*

Table 1

Decade	Male	Female	Total
0-10	None	None	None
10-20	3	2	5
20-30	20	14	34
30-40	24	20	44
40-50	24	13	37
50-60	11	10	21
60-70	6	1	7
70 & Over	1	1	2

It has been the observation of other clinicians that the number of females suffering from peptic ulcer exceed the males, but that has not been my experience. In this series there were 89 males and 61 females.

In considering my own cases of peptic ulcer, I have the habit of throwing the results into two categories in so far as the immediate results go, namely: the cures and the failures. I cannot see the logic of making a separate classification of those who are not wholly relieved of their symptoms at the conclusion of the period of treatment. It would appear that such cases ought to fall into the failure group. If all writers upon this phase of peptic ulcer were to eliminate this borderline group, our statistics in the future would have much more definite value.

Table 2 illustrates the ultimate results of the various types of treatment. From this table you will observe that of the men, 75 were cured and 14 were failures, giving a percentage of men cured as 84.26. Of the women, 54 were cured and 7 failures, giving a percentage of 88.52.

Table 2

Results	Males		Females	
	No.	%	No.	%
Cured	75	84.26	54	88.52
Failures	14	15.74	7	11.48

These percentages refer to ultimate and not immediate results.

Table 3 gives the types of treatment followed and their distribution between males and females. Seventy-two males were treated by diet alone. Of this number 59 were cured, giving a percentage of 81.9 for this type of treatment. There were 42 treated in like manner, with 34 cures, giving a percentage of 80.9. Duodenal alimentation was the type of treatment employed with ten males, all of whom were cured. There were 11 females treated in this way with 1 failure. Operative interference was resorted to for the relief of ulcer in 9 males, of whom 7 were cured, giving a percentage of 77.7. Six females were operated upon with 5 cured, giving a percentage of 83.33. The type of operation of choice was posterior gastroenterostomy.

From a study of this table we see that the best results in this series were obtained by duodenal alimentation with 95 per cent cured in both sexes. The average percentage of cures in diet and gastroenterostomy correspond very closely, the former being 81.4 and the latter yielding 80.5 per cent cures.

In this series of 150 cases, covering a period from 1905 to the present time, deaths from all causes as shown in *Table 4* were 13, or 8.6 per cent.

It may be interesting to note here that there were no deaths due to hematemesis, contrary to the reports from other clinicians. Although there were three cases of perforation, no deaths resulted therefrom. One patient died of carcinoma of the stomach fourteen years after discharge from treatment. There was one patient with primary carcinoma of the liver, who died eighteen years after discharge. There were 5 deaths from cardiovascularrenal disease, a percentage of 3.33. There were more deaths from this condition than from any other cause. One patient died of pulmonary tuberculosis and there was one suicide. Two died following operation from postoperative collapse and there were two deaths from causes which we were unable to discover.

Table 3

Treatment	Males		Females	
	Cures	Failures	Cures	Failures
Diet	59 81.9%	13 18.1%	34 80.9%	8 19.1%
Gastro-Enterostomy	7 77.7%	2 22.3%	5 83.3%	1 16.7%
Duodenal Alimentation	10 100%	0	10 90.9%	1 9.1%

Table 4

Deaths	Surgical		Medical		%
	M.	F.	M.	F.	M&F.
P.O. Collapse	-	2	-	-	1.33
Ca. Stomach	-	-	1	-	0.66
Cardio-Vasc.	-	-	3	2	3.33
Suicide	-	-	1	-	0.66
Pul. Tb.	-	-	1	-	0.66
Ca. Liver	-	-	1	-	0.66
Unknown	-	-	1	1	1.33

In this series there were 22 patients who claimed to have had a return of symptoms. From an analysis of the questionnaire received from these patients it is observed that they had suffered from the following disorders subsequent to their treatment for ulcer.

From *Table 5* we see that there were 3 cases, two men and one woman, who developed chole-

Table 5

Subsequent Disorders			
Return of Symptoms / Causes	M.	F.	%
Gall Bladder	2	1	2.0
Colitis	3	1	2.6
Failed Directions	5	1	4.0
Amebic Dysentery	1	-	0.66
Nephro-lithiasis	-	1	0.66
Unknown	4	3	4.6

cystitis. Of these 3 cases, cholecystectomy was done in one case two years following treatment, 1 case five years following treatment, and in the third case a diagnosis of duodenal ulcer and cholecystitis was made at the time of the initial study. A cholecystotomy was performed and the diagnosis of duodenal ulcer confirmed at operation. The patient remained symptom-free for fifteen years. Of these 22 cases, 6 admitted that they had made no attempt to follow dietetic instructions. Four of this number reported that they had been treated for colitis; in 7 cases we were unable to determine the cause of the return of symptoms. Attention has been called to the fact that the duration of symptoms prior to seeking relief has some influence on the ultimate outcome of treatment. In this connection it is interesting to note that in these 7 cases the symptons had extended from six to ten years. One patient had amebic dysentery and one had nephrolithiasis. It is fair to assume that in a certain percentage of these cases the symptoms complained of were due to these disorders rather than to the return of the ulcer.

From time to time attention has been called to the frequency of luetic infection in relation to the incidence of peptic ulcer. In my series not a patient gave a positive Wassermann reaction.

In recent years studies have been made showing the incidence of peptic ulcer in diabetes, but in this series it did not occur as a complication of ulcer, although careful study of the blood and urine was carried out in every case. However, within the past year we have had 3 cases of diabetes complicating ulcer. Under dietetic treatment ulcer symptoms disappeared along with the hyperglycemia and glycosuria. However, the time which has elapsed since the termination of the ulcer treatment is too short to determine the permanency of the cure of either condition.

It may not be inappropriate to state here that contrary to the practice of many clinicians, alkalies, in the treatment of peptic ulcer, were not employed to any great extent, light magnesia only being used. Early in my practice I formed the opinion that soda in any form administered to peptic ulcer cases not only did little good, but often seemed to retard healing. This experience gained in the early years of my practice has become a settled conviction after years of extensive experience with this type of digestive disorder.

And finally, permit me to affirm that from my own experience of thirty-five years in dealing with peptic ulcer, and from a study of this series and an exhaustive study of the literature bearing on this subject, I am convinced that individuals who have been cured of the subjective and objective symptoms of peptic ulcer, and have been free from symptoms for a period of five years, have not only as favorable a life expectancy, but are as comprehensively unimpaired for the carrying on of their accustomed vocation, as any similar group who have suffered any of the other serious illnesses which afflict the human body. It is to establish, insofar as it is humanly possible, this principle that I have deemed it appropriate to present to you this study.

THE ETIOLOGY AND TREATMENT OF FUNCTIONAL UTERINE BLEEDING*

By EMIL NOVAK, M.D.,
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When uterine bleeding is the result of a definite anatomic lesion in the reproductive apparatus, as is most often the case, its mechanism is usually easily understood. This is particularly true if the lesion be a destructive one, such as cancer, in which the actual opening up of blood vessels is the obvious cause.

On the other hand, when uterine bleeding occurs in patients whose reproductive organs are from an anatomic standpoint perfectly normal, its interpretation is by no means simple. The difficulties presented by this large group may be appreciated by the fact that they were, until recent years, quite commonly designated by the meaningless and unsatisfying term, idiopathic. Hundreds of papers have been written in past years in the effort to explain uterine bleeding of this sort as due to such factors as arteriosclerosis, insufficiency of the uterine musculature, or loss of elastic tissue in the uterine vessels. While all these factors may play an occasional role in the production of uterine bleeding, it is quite certain that they are not characteristically concerned in the etiology of bleeding in the great majority of the cases formerly

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*From the Gynecological Department, Johns Hopkins Medical School.

spoken of as idiopathic, and now more logically designated as functional.

It would be trite to emphasize that we cannot expect to explain deviations of a function unless we know something of its normal mechanism. And yet this is exactly what has in the past been attempted in the case of functional disorders of menstruation. The recent rapid development in our knowledge of the physiology of menstruation must make us realize on what insufficient foundation we had built our theories of former years. This is not to say that the physiology of the reproductive cycle is now a completed chapter, for much still remains obscure. Enough of the tangled skein has been unraveled, however, to give us a working concept of the mechanism of the normal cycle and to understand at least some of its abnormalities.

Functional uterine bleeding is one of the most common and most distressing of gynecological ailments. It occurs most often, in more than 50 per cent of all cases, in women at or near the menopausal epoch. Often (5 to 10 per cent) it is seen in girls at or near the age of puberty, or in the early adolescent years. The remaining 35 per cent or so of cases are encountered at any age during the reproductive era of life, for to this span the disorder is characteristically limited. It is true that in certain rare cases bleeding of exactly similar type is seen after, and perhaps many years after, the menopause. These exceptions, however, only prove the rule, for they represent instances of revivification of ovarian function under the influence of certain rare tumors of follicular or folliculoid type (granulosa-cell carcinoma).

The bleeding in cases of the functional type is most often of the hypermenorrheic or menorrhagic variety, though metrorrhagia is not uncommon. When the bleeding is of the periodic type, the intermenstrual periods are not infrequently much lengthened. It is easy to see how a period of amenorrhea of perhaps many weeks, followed by free uterine bleeding, may lead to the suspicion of early abortion. This is a frequent error in clinical diagnosis. Sterility is the rule with women suffering with functional bleeding.

The endometrium in cases of this type presents a very characteristic picture, spoken of as hyperplasia of the endometrium. It may be enormously overgrown and polypoid, so that the curette will bring away large masses which, in the woman of middle life, may well lead to the suspicion of cancer, if the operator is not versed in the naked eye distinction between the two conditions. In other cases, there is very little gross overgrowth, and in some cases, the endometrium may even be quite sparse.

Microscopically, however, the picture is essentially the same in almost all cases. I shall not discuss the histology of the condition except to say that the chief features are the so-called Swiss-cheese pattern of the glands (Novak) and the absence of any secretory activity in the glandular epithelium. For a fuller description of the pathologic changes, as well as for a more complete discussion of the clinical characteristics, reference may be made to several previous papers by the present writer.[1]

Emphasis may be placed, however, upon the ovarian changes, for these bear very directly upon the question of etiology, with which we are more directly concerned in this paper. The chief features are the characteristic absence of corpora lutea and the persistence of unruptured Graafian follicles. These findings would convey little significance were it not for the fact that the investigations of recent years have apparently crystallized into a fair working knowledge of the respective roles played by the follicle and the corpus luteum in the menstrual cycle.

The story is a long one[2] and can be touched upon on only one or two points most pertinent to the present discussion. Immediately after menstruation, the follicles begin to mature, and, for some unknown reason, one of them soon outstrips the others, to become the presiding ovum of the month. Under the influence of the follicle hormone the endometrium undergoes a slow developmental process (post-menstrual and early interval phases) during which no sign of secretory activity is to be seen. The follicle ruptures at about the thirteenth or fourteenth day of the cycle, with considerable individual variation, and after this the corpus luteum, in its turn, begins its march toward full maturity, to be attained at the onset of the next menstruation.

Under the influence of the corpus luteum hormone, or progestin, the development of the endometrium is carried still further, with a steadily increasing secretory activity in the gland epithelial cells. In other words, each menstrual cycle presents a follicle and a corpus luteum phase, the former non-secretory, the latter secretory.

In cases of functional hemorrhage, on the other hand, the follicle does not rupture, but continues to secrete its hormone long beyond the

normal period, so that there is an abnormally great follicular or non-secretory development of the endometrium. The failure of follicle rupture means an absence of the corpus luteum, and of course an absence of any secretory change in the uterine gland epithelium. It is the persistence and excess of the follicle stimulus which brings about the characteristic hyperplasia of the endometrium seen in such cases.

This is not to say that the endometrial hyperplasia is in itself the immediate cause of the bleeding, for this is probably not the case. Schroder,[3] Tietze[4] and others lay much stress upon the common finding of areas of localized necrosis of the uterine mucosa, although in our laboratory they have been observed much less frequently. It has always seemed to me that these areas, often very small, can hardly be looked upon as the source of the often extremely profuse bleeding exhibited by these patients, and that some local biological factor must be present in the endometrium, increasing the permeability of its blood vessels. This seems likely in view of the frequent gross intactness of the mucosa even when the bleeding has been profuse and prolonged.

Be this as it may, the underlying cause is of course the imbalance in the two ovarian hormones, as above stated. The endometrial lesion represents a physiological response to this disordered function just as the normal menstrual endometrial cycle represents the reaction to the orderly and sequential stimulus of the two ovarian secretions. Recent investigations, however, make it necessary to go back still further in our search for the ultimate cause of functional bleeding.

A brilliant chapter has been written by Smith and Engle,[5] Zondek and Aschheim,[6] and many other investigators in their demonstration of the governing role of the anterior pituitary lobe over the function of the ovary. This subject I have discussed more fully elsewhere,[2] and only one or two pertinent facts need be mentioned here. The anterior hypophysis appears to be even more fundamentally important in the sex cycle than are the gonads themselves. The cycle is abolished just as certainly by hypophysectomy as by castration. Follicle maturation is possible only under the influence of an anterior pituitary hormone, while a second hypophyseal hormone is essential for the luteinization changes characterizing corpus luteum formation. While these two governing or activating hypophyseal hormones are given different names by different investigators, their existence is now accepted by almost all the leading students of the subject. Zondek and Aschheim, whose investigations along this line have been most assiduous, give to the hormone governing follicle maturation the designation of Prolan A, while to that controlling luteinization they apply the name Prolan B.

The bearing of these facts upon the etiology, and also the treatment, of functional uterine bleeding seems obvious. The persistence of follicle growth, the failure of ovulation to occur, and the absence of corpora lutea all suggest that the underlying cause probably lies in a disordered balance between the motivating hormones in the anterior lobe of the hypophysis, and already there is accumulating a considerable amount of experimental evidence to support this view.

The application of the above facts to the management of functional hemorrhages can best be appreciated by referring to the older plans of treatment. The idiopathic hemorrhage of a former day, before the advent of radium or x-ray, was treated by rest, general hygienic measures, the administration of styptics of one sort or another, empiric and uniformly unsuccessful efforts at the ovarian organotherapy of the day, curettage, often repeatedly, with most often only temporary benefit, and, in the intractable cases, hysterectomy, even in young women.

The advent of radiotherapy constituted an advance of the first importance in the management of these trying cases. Without going into detail, the newer policies, developed after the advent of radium, and the ones still quite generally followed, might be summarized as follows:

(1) There are certain cases of functional hemorrhage of mild degree which correct themselves spontaneously. For example, many girls exhibit not only an irregularity, but also an excess of the function for a variable period after its inauguration. Often, however, after a period of months or sometimes several years, these patients fall into a normal menstrual swing, both as regards rhythm and amount.

The same statement may be made with regard to the manner of cessation of the function at the menopause. While normally the periods become less in amount as well as in frequency, there are not a few women who exhibit a tendency to flooding, and yet in whom the condition spontaneously clears up, with complete cessation of the function after a

variable period. While we, in our cancer programs, very properly teach women that flooding at the menopause is never normal, we should, in our own minds at least, recognize that in many cases the excessive bleeding is purely functional and that it may even disappear spontaneously. I need not urge that this fact should not put us off our guard to the extent of overlooking possible malignancy.

(2) When functional hemorrhage is so severe as to impel the patient to seek medical advice, the plan to be followed depends upon several factors, and especially upon the age of the patient and the severity of the bleeding. When the latter is alarming, curettage offers quickest immediate relief, whether the patient be young or old. At times it must be preceded by transfusion. When less severe, and when the patient is very young, so that the possibility of cancer need scarcely be considered, efforts at organotherapy are justifiable, although in the past almost always unsuccessful.

When the patient of middle life presents herself with bleeding for which no obvious anatomic cause can be found, curettage is clearly indicated, for it not only checks the bleeding, at least temporarily, but it establishes the diagnosis and eliminates cancer. Once the diagnosis is made by the finding of the characteristic endometrial hyperplasia, the further treatment of the patient at or near the menopausal age is simple. Here we have perhaps the most brilliant application of radiotherapy in the field of gynecology. Either radium or x-ray, in adequate dosage, yields virtually 100 per cent of cures by the induction of the menopause, for at this age the preservation of the reproductive function need rarely be considered.

(3) When bleeding recurs after curettage in young women, a serious problem arises, for here destruction of the reproductive function would be a calamity. The technic of radiotherapy has been so developed that, by careful small dosage, permanent sterilization can be avoided as a rule, with only temporary cessation of the function. When it is re-established, it may be normal or it may again be excessive, necessitating further radiation. Unfortunately, however, there are some women who are extremely sensitive to the effect of radium, so that even very light dosage may bring about permanent amenorrhea and sterility. Numerous instances of this sort have been recorded, so that almost all gynecologists and radiotherapeutists are agreed that radiotherapy in young women should be only a last resort.

Here the only alternative in the past has been a repetition of the curettage, perhaps several times, depending on the frequency and severity of the recurrences which are so common. When the problem is fairly presented to the patient, or, in the case of young girls, to their parents, almost always the choice will be a repetition of the curettage rather than radiotherapy, and this has been my own practice in the past in all except the occasional case of unusual severity or pertinacity.

It is this group of cases, that is, functional hemorrhage in young individuals, which has constituted the most serious problem, in fact, one of the most trying problems in gynecological practice. For this reason, we have been extremely gratified at the excellent results of a plan of treatment which we have been developing within the past year. I refer to the use of an anterior pituitary luteinizing substance obtained from the urine of pregnant women, and supplied to us through the courtesy of the Department of Experimental Medicine of Parke, Davis & Co. The rationale, technic and results of this plan are fully discussed in a paper by Hurd and myself[7] in the current number of the *American Journal of Obstetrics and Gynecology*, and to this the reader is referred.

Suffice it to say that the idea behind this treatment was to activate luteinization in the ovary by the injection of the anterior pituitary luteinizing hormone. As the lack of lutein tissue is the chief ovarian factor in this disorder, this seemed rational. The cessation of the bleeding in many of our cases, however, was so prompt that it could hardly have been brought about by ovarian luteinization, for the latter would require a considerable time, probably a number of days. We believe, therefore, that the effect is produced, in a parallel fashion, upon the bleeding factor demonstrated by Hartman, Firor and Geiling[8] as produced in the anterior pituitary. These physiological considerations we have elaborated upon in the paper above alluded to.

We have employed this plan in the treatment of more than 100 cases of functional hemorrhage, many of them of the recurring type, with often a history of one or more curettements. Most of the patients have been young, so that radiotherapy would have been undesirable. While not by any means infallible, the bleeding, in all except a small proportion, has been checked by the intramuscular injection of usually 200 rat units of this preparation for

from two to five successive days. In bleeding of the periodic type, the injections are begun with the onset of the period. In cases of the metrorrhagic variety, the treatment is begun as soon as the patient comes under observation. There are no ill effects from this treatment except the local reaction, which is usually not severe, and which will probably be eliminated as the preparation is perfected.

Unfortunately, the effect of treatment appears to be temporary, that is, to be limited to the particular bleeding attack. Very few patients, however, would object to a few injections each month if operation or radiotherapy can be thus avoided, as it generally can. This is particularly true in view of the undoubted tendency in very many of these cases toward readjustment of the endocrine balance, so that after a longer or shorter period menstruation may again become normal. If the patient can be tided over the period of endocrine upset by some such simple plan as ours, without operation or radiotherapy, the boon is a real one. We are so convinced of the value of this procedure that we would not think of resorting to more radical measures in young patients until this plan of organotherapy has been tried.

SUMMARY

(1) Functional uterine bleeding, an exceedingly common disorder, is due to a relative increase and persistence of the ovarian follicle hormone, with an absence of the corpus luteum secretion. This ovarian dysfunction, in turn, is almost surely due to a disordered balance between the follicle ripening (prolan A) and luteinization (prolan B) hormones of the anterior hypophysis.

(2) The treatment of this disorder in women at or near the menopause, when the preservation of the reproductive function is rarely an important consideration, is simple enough, once the diagnosis has been made by curettage and microscopic examination. Either radium or the x-ray yields uniformly good results through the induction of the artificial menopause.

(3) The treatment of functional hemorrhage in young women, in whom it would be wrong to endanger reproductiveness unless absolutely necessary, is a much more difficult problem, and radiotherapy should be resorted to only in cases which persistently resist all other measures. A very valuable adjuvant in this group, we are convinced, is the intramuscular administration of the anterior pituitary luteinizing hormone. This plan is a rational one on physiological grounds, and in our hands has yielded very gratifying results. It will in many cases obviate a resort to repeated curettage or radiotherapy.

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DISCUSSION (*Abstract*)

Dr. C. Jeff Miller, New Orleans, La.—For twenty-five years we have listened to theoretical discussions of the treatment of uterine hemorrhage by endocrine therapy. Today Dr. Novak has offered us facts, facts proven first in the laboratory and then substantiated in the clinic. For many years he has contended that endocrine therapy could not be expected to achieve results until it was given with the idea of reproducing the processes of the normal menstrual cycle, and the recent investigations in the field of both physiology and endocrinology have established the entire correctness of his attitude.

In the past we have been more or less helpless in the management of functional hemorrhage unless we invoked methods which destroyed function forever. We no longer resort to hysterectomy, except in the exceptional case, but we do use radium in the treatment of menopausal bleeding of the functional type and we have in it a very powerful and a very helpful agent. But for the bleeding of young girls and the bleeding of young women, in whom destruction of function would be truly a catastrophe, we have been obliged to rely chiefly upon curettage. We realized, when we used it, that its use was irrational and illogical; we realized that we were not striking at the true source of the hemorrhage; we realized that in many cases the bleeding would return. We did, however, cure some patients by this method, though I think nobody can explain why we did, and we tided many of them over their trouble, perhaps by repeating the procedure, until nature took a hand and restored normal function or until the menopausal years had come and the destruction of function by the use of radium was no longer a vital matter.

Now, however, the treatment of functional bleeding has passed beyond the tentative stage. Biochemist, physiologist and clinician have joined hands, and the result is the beautiful piece of work which Dr. Novak has just presented to us. The ovarian substances which we prescribed in the past by the ton and without reason were ineffective as they could not fail to be; they were valueless from the standpoint of potency, and they were shots in the dark from the clinical aspect. The new method attacks the disease, not in its secondary manifestation in the ovary, but in its primary manifestation in the anterior pituitary gland, and when once the extract is made generally available, the control of functional bleeding will be both rational and possible.

Two points I should like to make. The first is to emphasize a point which has already been made, that correct diagnosis is the essence of correct treatment, and that diagnostic curettage is particularly important in adult women or in women in the cancer years to rule out the possibility of malignancy. The second point is the value of transfusion in the management of functional bleeding in young girls. I stumbled on this measure by accident some time before it got into the general literature by using it as an emergency treatment, and found, to my surprise, that in a very fair percentage of cases it wrought a cure, though why it should I could not undertake to say.

Dr. John C. Burch, Nashville, Tenn.—The material presented by Dr. Novak is not theory, but fact. The pathological picture of Swiss-cheese hyperplasia can be produced experimentally in a number of ways. Some time ago my colleagues, Drs. Cunningham, Wolfe, Williams and I presented some work on the etiology of this condition. Recently we have obtained some more typical pictures in our experimental animals. A castrated rat which had received small doses of an estrous producing hormone, amniotin, shows a uterus typical of human hyperplasia in all of its pathological details. The Swiss-cheese dilatation is exceptionally well shown.

The luteinizing hormone is excreted in the urine of pregnancy. It is assumed to be of anterior hypophyseal origin, because fresh anterior hypophysis and pregnant urine produce the same changes in the reproductive tract of mice and rats. A substance somewhat similar is found in the placenta. It is as yet uncertain whether the luteinizing hormone of the urine, the anterior pituitary substance of the placenta, and hormone of the anterior hypophysis are all the same or are all different. Good results in the treatment of functional bleeding have been obtained from alcoholic extracts of the placenta and from the luteinizing hormone. The luteinizing hormone is now in preparation by several of the larger biological houses and is on clinical trial. In several instances we have used one of these preparations. Prior to their appearance we had been using small amounts of pregnant blood which contains the luteinizing hormone. This has been used frequently abroad by Ehrhardt and others. Our results have been so good that we feel justified in continuing the procedure and in recommending it to others. Ten cubic centimeters of blood are taken from the cubital vein of a pregnant woman and immediately injected into the buttock of the patient. No anticoagulants are used. The donor is previously examined for lues. Recently we have tried sealing the tubes and preserving the blood in a steri'e condition. When the tube is broken the serum is collected and injected. This has so far been satisfactory, but has been used in only a small series. Most of these patients have been given a dose of pregnant blood on three consecutive days. At the end of this time the bleeding has usually ceased and the patients generally return to a regular cycle.

Dr. P. B. Salatich, New Orleans, La.—I have been able to arrest a case of severe bleeding, in which the patient was almost exsanguinated, by transfusion. The patient had no subsequent trouble.

Dr. Novak (closing).—Dr. Miller spoke of the practicability of the employment of the luteinizing hormone in cases of functional hemorrhage. The treatment is already a practicable one, though admittedly new and still very imperfect. Our chief difficulty now is that the material often produces unpleasant reactions, usually only local, but occasionally even systemic. We have been assured by Parke, Davis & Co., however, that this unpleasant feature will probably be eliminable before long. It is hoped that the material will soon be placed on the market. The blood of pregnant women also contains these luteinizing substances, as Dr. Schwarz has pointed out, although the use of pregnant serum is not so convenient as the use of an assayed product such as ours. We are hopeful that it may soon be possible to prepare even more potent substances from the gland tissue itself.

The possibility of origin of these supposed pituitary substances in the placenta is now receiving wide attention, especially in the German literature. A rather sharp controversy on this point has been carried on between Zondek and Phillip during the past year. There is also still some doubt as to whether the prolan in the urine of pregnant women really represents the sex hormones of the pituitary, although there can be no doubt of its luteinizing properties, in at least some species of animal.

MENTAL REACTIONS ASSOCIATED WITH THE MENOPAUSE*

By ELEANORA B. SAUNDERS, M.D.,
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Tradition and superstition offer explanation for many manifestations of human life not otherwise understood. It is true that our instinctive demands and activities are at times a puzzle and even a source of awe to many of us. But submission with little attempt at explanation or correction is nowhere more prevalent than in the phenomena, both normal and abnormal, associated with the sex life of woman. During no age may the evidences of acceptance and repudiation, adjustment and deviation be more frankly expressed than in the complaints at the involution period. Menstruation is closely associated in the minds of many with health and disease and is often accepted as both cause and effect of mental and physical phenomena entirely separate and apart. Emotional and neurotic manifestations are commonly viewed as a necessary concomitant to the menopause and too much is attributed to this period *per se*. The "change of life," then, often becomes a synonym for doubts and misgivings and any emotional disturbance may be ascribed to this biological phenomenon which is accepted as adequate cause for any and all ills from thirty-five to sixty. Indeed, it is a prevalent belief that the fullness

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*From the Sheppard and Enoch Pratt Hospital.

and completeness of young womanhood depend on the menstrual activity. Women have been known to entertain grave doubts in regard to the state of their health if there is the presence of amenorrhea arising from causes other than pregnancy. They are disposed to attribute any mental or physical discomfort to the absence of menstruation; and with its failure in later years they experience a feeling of loss manifested in a renunciation of all satisfactions, the more strictly sexual as well as the more frankly asexual pleasures. Many somewhat fallacious beliefs accepted by normal women easily form a topic of preoccupation for those in whom there are doubts and misgivings arising from vague discomforts and from feelings and thoughts foreign to their usually accepted standards.

Adjustment on a frank reality basis, even in the most normal, at this period of life is that of loss, reproductive, possibly economic. Retrograde changes are to be expected, but the attitude of loss and of renunciation of satisfactions in life with a general feeling of incompetence are destructive in their potentialities. Neglect arising from too implicit acceptance of invalidism at the menopause may result in discouragement and lack of constructive physical or mental intervention. The involution is a period of accommodation to loss, to a waning capacity which requires revaluation of assets for the healthy as well as for the neurotic. It is a difficult period of life, to be sure; but of much more importance is the reaction of woman to it. She often comments on a feeling of being less attractive and of being less able to sublimate energies which sometimes apparently flare up anew with the approaching loss of the function of reproduction. She may even be thrown back to a lower level of adjustment, to discontent with her partner or the reverse; or she may become self-centered, auto-erotic and narcissistic. These attitudes are merely an accumulation of reactions throughout life, a reflection of early adjustments. Behavior responses at the menopause may be a reactivation or an extension of emotional instability which is present at puberty. Invalidism of the involution period may derive motivation from fancies and trends which were alive in youth, but which lay dormant during the more successful productive years.

The menopause is not, in itself, the sole cause of involutional mental instability, but the underlying tendencies, the individual reaction would seem to be the more potent factor. One must look beyond the mere menstrual cessation for causes of emotional disability at this time of life. Certainly, eccentric personality traits are less well accommodated with this biological failure. Sex-preoccupation is not woman's sole mental content, but it is a big factor in the adjustment to loss of the specific feminine function. With this loss she turns, in healthy states, to sublimations through professional and civic interests approaching more closely the masculine because both before and after the menstrual life of woman her adjustment may be on a plane of activities which is not specifically reproductive.

While there is no individual response, the mental symptoms arising as a result of compromises and failures may assume neurotic or psychotic formation. The so-called neurotic traits are expressed in tensions, fears, and misgivings. Anxiety states and depressive manifestations are the feeling realizations which usually accompany the initial phases of emotional stress incident to the declining feminine function. In the post-climacteric period there is a tendency to the intellectualization of the feeling problem, to obsessive or paranoid trends, and sometimes to melancholy reactions. The types of psychoses which occur most frequently in late life are: (1) the neurotic or eccentric personalities who, having made a precarious adjustment in earlier life, at the involution period take on a more sinister symptom formation as seen in atypical depressive and projection states. These lead frequently, as the case develops, into terminal dilapidation of habit and of thought formation with great loss of acumen; (2) the late developments or recrudescence in a prolonged illness of schizophrenic nature; (3) the organic-arteriosclerotic or senile manifestations; and (4) the affective or manic depressive.

Stresses or conflict manifested as difficult character traits of early years may ripen at this period of life into a more profound involutional affection. The meticulous housekeeper, disturbed by disorder and conscientious in detail with a drive to maintain ideals of cleanliness and orderliness, not infrequently becomes more duty-driven and exacting. Or the emotional, somewhat hysterical type, often irascible and difficult with servants, who fluctuates between feverish activity and withdrawal to invalidism when thwarted, may become dramatic and disposed to outbursts of temper. A tendency in early years to uneasiness about her personal comfort and the condition of her health may

gradually mature in late life with the formation of poorly organized delusional trends, transient excitements, and tense depressive states with compulsive and suicidal behavior or invalid (conversion) phenomena. Patients of this type are frequently crafty and difficult in problems of food intake and general care. In many instances the physical condition suffers through excessive activity due to tenseness and sometimes to insufficient food and rest.

General physical debility and coincidental somatic abnormality make an evaluation of psychogenic and physiological factors difficult. Extensive medical skill and resourcefulness have not infrequently been expended on a somatic syndrome of psychogenic origin. Desexualizing surgical intervention has many times satisfied a feeling of guilt through the suffering and privation and has given opportunity for projection of responsibility to the physician. Following professional advice given and medical or surgical treatment undertaken, a patient may continue without feeling of guilt a habit previously practised with much self-censure, or she may react with great self-incrimination and feel that because of the indulgence she has been punished by the loss of the organ with which she has sinned; and in a late life depression she may feel that she is condemned and as a result must endure eternal punishment. In this connection mention may be made of the spinster neurotic type with attachment for a brother or near relative, who for a period of years has had an invalid reaction expressed in conversion symptoms, and who, in late life, is prone to an accession of depression with frenzy periods during which there may be impulsive attempts at suicide. Chronic semi-invalids may for years have episodes of depression with any additional stress and the hypochondriacal habit may terminate in a late life depression.

Acute late life disturbance may be the culmination of schizophrenic trends which may have matured through gradual change over a period of fifteen to twenty years, during which time the patient may become more seclusive, duty-driven, and sensitive. A middle-aged woman complained of somatic discomfort, "indigestion," for fifteen years and became much more distressed following the death of her father two years before she was admitted to a mental hospital. An operation on the gastrointestinal tract was complicated by delusions that patients in the surgical institution were detectives whose duty it was to watch her. There were many ideas of self-reference and unreality. While in a convalescing hospital she spoke of impregnation when she was asleep and projected this delusion to a workman. Ruminations of pregnancy "with resulting body change" and ideas of persecution were the presenting features of her illness. It would seem in this case that the delusional content directed the mood variation. With the passing of time she became more indifferent and withdrawn, more under the direction of erotic preoccupations and of somatic and persecutory delusions, with corresponding behavior alteration.

The more frankly paranoid adjustment, striking in its apparent sudden departure from the former conjugal content of a dutiful wife, is demonstrated in the formation of doubts, misgivings and projections which she may direct toward her previously beloved husband. This aggressive attitude may also be seen in the unmarried woman who, having made an adjustment on a professional basis, at the menopause period expresses as a fact the wishes and strivings which she formerly entertained on a more autistic level. The delusions arising from her expectations and her instinctive demands result in aggressive threats which are sometimes potentially homicidal to "enforce her rights."

Age likelihood has long been accepted as a factor in causation of disease, both physical and mental. In neither type of sickness, however, do we find a specific reaction, but rather an individual response to situations. Manifestations of behavior at adolescence or at late life present no response peculiar to those years alone. Advanced old age may give evidence of childish traits of body or mind in reaction to illness or to stress, and prolonged physical sickness even in robust years may be complicated by regressive behavior characterized as infantile. While we know that symptoms of mental illness may develop at almost any age, epochs of life offer an added tendency: puberty with schizophrenia and maturity with arteriosclerotic changes. In both of these affections the individual trend or set of life is a big factor: in the one, the chief expression; in the other, the freeing of trends of personality which have been developing since early years. Biological failure is usually thought to precede the organic; however, the tissue change may be the earlier manifestation in the arteriosclerotic types. Here the lessened integration of personality permits an easy outcropping of depressive, of projection or of anxiety traits previously well covered. The

irritability, the labile mood, and the memory lapses, while classic, are often covered by symptoms indicating compensations and inherent inferiorities which direct the mental trends. It is these individual factors which motivate the formation of depressive, manic, paranoid, or invalid tendencies. The organic syndrome may occur coincidental with the illness of psychogenic origin or may, by its disability, precipitate a later depressive reaction arising subsequent to its onset.

Apprehensions and fears of cancer or syphilis and conversion-like prodromal phenomena of late life psychoses, especially depressive reactions, are the most frequent associations of the physical and the mental in thought or behavior manifestation of patients at this age. Psychoses of the late life period may be preceded by prolonged "ill health" manifested in somatic complaints. Following several years of "physical sickness," conversion, or neurotic symptoms, there may be a somewhat acute depression breaking through into a frenzy of anxiety and apprehension. Often with added stress resulting from disappointment, incurable disease of a partner, or loss of property, patients may begin to complain of gastrointestinal discomfort, "neuritis," or conversion "hysterical" symptoms which seem to be a reaction formation adequate to support the stress for a period of several years or even longer. Then, with greater or with added hardships, there may be a collapse into a frank depressive reaction. With development of depression, there is usually a lessening of complaints and of physical symptoms. Hypochondria, irritability and deepening depression are often frankly associated with a disturbance of the love object. In cases of this type there may be complaints of physical illness with many attempts at relief through medicines and Christian Science; but the patient tends to become at the involution more depressed and agitated and may develop into a frenzy arising from unreality notions. Patients of this type may continue for months with furious agitation of a continuous nature which may result in great emaciation and much exhaustion.

Depression, the most frequent manifestation of late life failure at adjustment, with its somatic notions, its unreality feelings, and its projections, is more complex than the affective illness in early life. These gloomy, agitated patients maintain an attitude of sadness and of dejection with fluctuations during the day and with many remissions of longer or shorter time during the course of an illness which must be reckoned in months and sometimes in years before recovery. While the acute onset usually makes for a more favorable outlook, this single factor does not alter unconditionally the course of the late life mental illness, because from the abrupt setting in of symptoms a chronic illness may develop and patients with insidious onset may recover. The real experiences causing sadness in many patients suffering with late life depression make it difficult for them to create a setting of enthusiasm or even of interest. Some patients in response to the actual conditions of life make an adjustment of a very fragile character in which depression seems to be a reaction of failure to attain adequate satisfactions. While the feeling tone of the patient suffering with affective illness is of utmost importance in evaluating the content, the situation in life, social and economic, the problem of actual age, and the troublesome question of what life holds out for her are factors to be considered in the prospect of recovery.

As a result of observations over a prolonged period of time of a group of patients with emotional disturbances at the menopause it appears that we may conclude, first, that too ready acceptance of tradition and superstition make it easy to accept invalidism as a necessary concomitant of the menopause; second, that while the involution is a period of life in which biological failure is to be expected, a more competent emotional adaptation may be made, not on a basis of renunciation of satisfactions in life, but as a result of a readjustment of the balancing factor essential for a healthy mental state at the menopause.

DISCUSSION (*Abstract*)

Dr. David C. Wilson, University, Va.—Dr. Saunders has presented in a splendid fashion the underlying mechanisms producing the preoccupations and overt acts of a large group of personality disorders. She emphasizes the fact that it is not the biological change *per se* which produces these disorders, but habits of thinking both racial and individual, grouped about this period of life, as well as the economic, financial and libidinous alterations either assumed or real which accompany it. Next the response is classified and the reactions divided into neurotic and psychotic. The anxiety states with depression are classified as neuroses and then as psychoses, first the neurotic type making a difficult adjustment that dilapidates under the mental and physical strain; second, a flight into schizophrenic chronic invalidism; third, an organic arteriosclerotic or senile manifestation; and fourth, an affective or manic-depressive reaction type.

The classification is repeated as insight regarding the variety of response to be expected is most helpful as, although in this reaction type as in all other mental reactions, the personality and its intimate make-up color the picture, in the understanding of this most confusing group segregation into smaller categories is necessary. The greatest contribution of the paper, however, seems to me to be that the menopause or the presenium is not one event, but a strain lasting over many years, and that the individual may be able to adjust on one level for quite some time and then finally gradually fail and regress and become more and more submerged.

As the writer indicates, one individual may show many phenomena, conversion tendencies, obsessive, paranoid, manic or depressive trends as the collapsing personality liberates suppressed material, so that this liberation may, for many years, appear as simple hypochondriasis with troublesome somatic complaints; then later, as the process continues, engulf the whole individual, disclosing the gravity of the underlying mental situation. The care, the treatment and, above all, the understanding of these involutional reactions is most difficult, therefore this enlightening presentation is most welcome.

Dr. Walter J. Otis, New Orleans, La.—The different symptoms that Dr. Saunders has given us may be classed as menopause neurosis or psychosis, depending on the stage reached. As little reference as possible to the sex organs should be made at these critical times. Mental hygiene should be stressed.

The question of sexual hygiene should be presented intelligently.

Dr. Isham Kimbell, Alexandria, La.—Some one has placed the dangerous age for man at the third trimester, from 40 to 45 years of age. At this time he is vigorous, sliding gracefully and commandingly through his declining years. Woman is no less vigorous at this time of life, but she is brought face to face with a physical fact, the menopause.

The neurotic and unstable woman tries to elicit sympathy, and often expresses her conflicts in symptoms referable to that part of her body which is now, at the involutional period of her life, commanding much of her attention. Thus we have hot flushes, pelvic pain, backache, weakness, and a host of other somatic complaints.

Those of us who have had opportunity to make observations of patients during the involution, or, rather, during that period of life in which biological failure is to be expected, must agree with Dr. Saunders that "the reaction to this loss is not to be accepted as renunciation of all life satisfactions;" invalidism is not a necessary concomitant of the menopause. It is unquestionably true that woman is often confronted with difficult situations at this period in her life, which are productive of abnormal emotional reactions; but a study of the patient will reveal traits of personality which are the etiological factors in her maladjustment.

Many of us believe that there are no other medical specialties which touch so closely the field of psychiatry as do obstetrics and gynecology. There is much need for proper understanding of the mental reactions of the woman who comes to the gynecologist for relief of a pelvic disorder, or of the pregnant woman who comes to the obstetrician.

At one time there was widely prevalent the idea that any abnormality in the pelvis, especially when seen at the menopause, retroversions, a too freely movable uterus, simple infection and laceration of the cervix, slightly relaxed vaginal outlet, caused a large part of the nervous disturbances or neurotic manifestations in women, and when these were found, operative interference was immediately instituted.

The best clinics and the well prepared gynecologists no longer operate upon neurotic women with minor pelvic disorders.

The menopause is not the sole cause of involutional mental instability. We must look beyond the mere menstrual cessation and into the individual mental reaction for the causes of emotional disabilities.

Sex preoccupation is a big factor at this time of adjustment to loss of the specific feminine function, and its loss brings about sublimations through professional and civic interests approaching more closely to masculine activities.

The correct evaluation of the neurotic or psychotic manifestations at this period is sometimes quite difficult. Dr. Saunders' classification is good: we do have the atypical depressive and projection states leading to terminal dilapidation of habit and thought formation with loss of acumen; the late developments of a schizophrenic nature; the senile manifestations associated with the organic arteriosclerosis; the affective or manic depressive.

Schizophrenic trends maturing later in life are not difficult of recognition; the numerous somatic complaints without organic basis which these patients frequently exhibit, the many clinics and physicians they have visited, the multiplicity of diagnoses they have received, all tend to make us search carefully for evidences of maladjustment in early life, for stresses and conflicts, evidence of difficult individual character traits.

The anxiety states, which we now recognize as dependent upon frustrated, or, rather, dammed-up libido, frequently appear at this time as feeling realizations accompanying the initial phase of emotional stress incident to the declining feminine function. Anxiety frequently goes along with hypochondriacal preoccupations, but the hypochondriac does not attach her libido to external objects. She is auto-erotic; her libido is devoted entirely to the various parts of her anatomy.

The emotional or hysterical type is interesting, dramatic, fluctuating between feverish activity and invalidism, exhibiting many conversion symptoms, showing a tendency to invest various parts of her body with the energy derived from latent sex impulses. These maladjusted individuals frequently seen at this period of life may, because of an accentuation of difficult individual character traits recognized at puberty as emotional instability, accelerate, retard or misdirect the social wheel; their manifestations which were recognized at first as severe neuroses may become true psychoses. Regression may become evident with loss of insight, attempts at self-destruction because of depression and intolerable situations, there may be projection mechanisms, divorce from reality, phantasy life, dissociation between object and affect, and between intellect and emotion.

THE USE OF THYMOPHYSIN AND SIMILAR SUBSTANCES IN LABOR*

By RICHARD PADDOCK, M.D.,
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Since 1926, when the reports of Temesvary, and others using thymophysin, were published in a number of European journals, this and similar preparations have received considerable attention. The remarkable claims made for thymophysin by numerous European authorities quite properly turned the eyes of interested obstetricians upon this subject. When thymophysin and similar preparations became easily available in this country, many enthusiastic practitioners obtained these preparations and began their clinical application.

From the rather extensive use of these thymus-pituitary preparations, there have reached the literature numerous reports of series of cases in which these substances were used. With few exceptions, most of the comments have been highly favorable and bear out the claims made in the earlier reports.

Let us consider the substances in question and seek the causes of their rather widespread popularity. The claims made for the material seem to originate with the work of Temesvary. His studies with various organ extracts brought him to the conclusion that thymus extract increases the uterine tone and intensity of contractions, which remain rhythmical. When thymus extract is combined with pituitrin the uterine contractions become greatly increased but rhythmical. In addition to the intensified rhythmical contractions, the thymus extract is also said to relax the cervical muscle, which allows the cervix to dilate and to offer little or no unsafe resistance when the intensified contractions effect the expulsion of the child. On account of these ascribed attributes, the substances are recommended for use in the first stage of labor.

Shortly after the introduction of pituitary substance for use in labor, its dangers were pointed out. As a result of the abuse of pituitary drugs, extreme caution in their use was advised. Accurate methods of standardization were devised so that such preparations might be used with safety. In spite of the careful teaching of caution, and the numerous unfortunate reports, pituitary extracts have been and are still being misused to a very great extent. The rapid termination of labor following the injection of pituitrin seems to be the sole indication for the drug in the minds of many practitioners. It is not unusual to hear of practitioners who use ampoule doses of pituitary extract as soon as they reach a patient in labor.

Now when a preparation so highly recommended as thymophysin is available, when the internationally known leaders in European clinics reported with favor series totaling hundreds of cases in which thymophysin was used, it is no wonder that the busy practitioner was willing to substitute this new preparation for the old pituitary extracts. The addition of thymus extract to pituitary extract seemed to break the spell that held cautious obstetricians from the indiscriminate use of pituitary preparations.

American manufacturers were alert to the possibilities of this new preparation and quickly made it available. The literature sent out to the profession is full of glowing accounts of numerous case reports by well known authorities who have used thymus-pituitary substances.

The original work of Temesvary was based on a considerable amount of pharmacological work with organ extracts. Certain results obtained by him appeared favorable for clinical application and were accepted by him as more or less conclusive. Certain errors in Temesvary's conclusions were pointed out at the time by a physiologist, Herman Knaus, but they were seemingly inundated by the tide of favorable opinion that clinical reports brought forth. In all of the earlier reports, no unfavorable results were reported, so that any possible harmful effects were seemingly overlooked.

A few dissenting voices were raised, but the majority seemed quite favorable. Dr. Joseph B. DeLee, in an open letter in the *Journal of the American Medical Association,* seemed to be the first in this country to take an adverse stand, based on a purely obstetrical viewpoint. In recent months certain critical reports have appeared in the literature of cases of ruptured uteri and of asphyxia and birth injury of the newly born attributable to thymophysin. Nelson, in the *Journal of the American Medical Association* of January 31, 1931, presents the pharmacological tests of thymophysin in a most comprehensive manner. His conclusions are adverse to those on which the theory of thymophysin is

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based. At the same time Rucker draws attention, by bedside laboratory methods and observations, to adverse effect with thymophysin. Greenhill, in his obstetrical review published in July, 1930, compared results obtained with thymophysin and with pituitary extract alone. It is interesting to note that some of the literature sent out by one of the manufacturers quotes Greenhill on maternal mortality to sustain the arguments in favor of thymus and pituitary extract.

The pharmacological effect of thymus extract is not well defined. On account of the rather indefinite activity of the substance, standardization of the extract is difficult. In the literature prepared by the manufacturers, details concerning the thymus extract are omitted.

It was our desire to observe the effect of thymus and pituitary extract on our own cases and thus evaluate clinical results to our own satisfaction. With the cooperation of the residents on obstetrics, namely: Drs. Soule, Wegner, Hobbs, Rollins, and O'Neill, a series of observations were made. Four preparations of thymus and pituitary extract were used. Most of these drugs were obtained through the kindness of the manufacturers, while some were purchased on the market. Three of the preparations were in trade packages, while one preparation was not on the market, but was put up for experimental purposes only. They will be referred to as A, B, C, and D, respectively. In the series there were 134 cases, divided as follows: A 59, B 43, C 11, and D 21.

	Primipara	Multipara
A	31	28
B	20	23
C	2	9
D	13	8

In our first observations to familiarize ourselves with the effect and dosage, thirty cases were followed using thymophysin in the dose recommended in the literature, 0.5 to 1 c. c. The results obtained were in general in accordance with those described in the literature. Labor usually terminated in three to four hours after the first injection in primipara, and in thirty to forty minutes in most cases in multipara. For conservative obstetrics, it was concluded that the doses were too large and the effect too pronounced. The contractions produced were too strong in many cases to be considered safe. The difference between the first effectual contractions and tetanic contractions seemed only a matter of individual definition.

The dosage followed in the succeeding cases was in most instances less than the recommended dose. The cases were selected where no unfavorable results from any cause might be obtained. The basis for selection in a large number of the cases was slow progress without evident dystocia. Usually some degree of inertia was present where the use of pituitary preparation in small doses might be considered. Usually the contractions were only fair and irregular when the case was selected for observation. No cases having strong uterine contractions were selected.

Effect of the Drugs.—After the administration of the drug, an appreciable effect was noticed in almost all of the cases. Only one case in active labor showed no effect. There were no progressive effects in four attempts at induction at term. These four patients went into spontaneous labor at a later date and delivered without difficulty.

The administration of the drug was intramuscular in every case. The initial dose in most cases was given with the patient in the first stage of labor, with appreciable cervical dilatation. In 19 cases, the initial dose was given during the second stage. If the nature of the contractions later became such that another dose was indicated, it was repeated. The shortest time interval between injections was forty minutes. The most injections in any case were five.

The first effect after injection was usually noticed in three to five minutes. During the first fifteen minutes the contractions were greatly intensified and less regular, but more frequent, with an increase in muscle tone anywhere from a mild increase to tetanic contractions. Usually after the first fifteen minutes the contractions became more regular.

In three cases severe tetanic contractions were noted. Two of these were with the recommended dose of one preparation and one with a two minim dose of another preparation.

As the size of the dose was reduced, the time of delivery after the injection was increased. Where the cervix was observed for change in tone or resistance, there was no evidence that any change took place as a result of a specific action of the drug. The force of the contractions produced seemed to account for any change observed in the cervix.

The blood pressure change was negligible following the injections. During the first few min-

utes of the initial effect the average increase was less than 10 mm.

The condition of the child at birth was good in most cases. In 16 cases some methods of resuscitation were considered necessary. Possibly too large doses were used in some of these cases where the child showed some asphyxia. There were no fetal deaths. All the children were discharged living, without signs of birth injury.

The labors were managed in the same way as equal numbers in which the patients did not receive thymus and pituitary substances. Analgesia of some sort was attempted in all cases.

76 received morphine hyoscine
7 received quinetherol
10 received allylisopropyl barbituric acid
3 received sodium amytal

A number received nitrous oxide with the contraction.

For delivery most of the patients had chloroform anesthesia. Some had gas or ether.

The character of the delivery was the same as in any equal number of cases. Most of the primipara and a few of the multipara were delivered by perineal forceps and episiotomy. A mid forceps and low forceps were performed as indicated on a few cases.

The character of the third stage was normal in most instances. No cervical lacerations were noted and no cervical repairs were done. The tone of the uterus following delivery was very good in most cases. None of the patients experienced any hemorrhage. There were no maternal deaths nor serious puerperal complications.

CONCLUSIONS

(1) All of the preparations of thymus and pituitary extract used exerted a definite stimulatory effect on the muscle of the uterus in labor not appreciably different from that from small doses of pituitary extract alone.

(2) The doses recommended by the manufacturers are far too large to be used with safety.

(3) Small doses are less effectual in most instances than equal volume doses of standard pituitary preparations.

(4) Observations showed no evidence to support the assertion that the addition of the thymus extract in any way lessened the resistance of the cervix in labor.

(5) There is no more indication for thymus and pituitary extract in normal labor than there is for pituitary preparation in normal labor.

(6) Some of the claims of the manufacturers of these preparations are misleading and should be modified to comply with the facts.

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DISCUSSION (*Abstract*)

Dr. W. T. Pride, Memphis, Tenn.—The abuse of such preparations as thymophysin and pituitrin is too prevalent. My experience with thymophysin has been very limited, because I use analgesics rather than contractants. We were told by good chemists that the thymus extract is inert so far as uterine stimulation goes. We tried thymophysin in the Memphis General Hospital and it caused such violent uterine contractions that chloroform had to be given for fear of uterine rupture.

Dr. Temesvary claims for it rhythmic contractions, but these are too violent for my service.

Dr. James R. McCord, Atlanta, Ga.—All of these cases were carefully selected. Dr. Paddock was trying to observe the therapeutic effects of thymophysin rather than to shorten labor.

I am always suspicious of any drug or procedure upon which the comments are always highly favorable. Dr. DeLee some time ago called our attention to the fact that, at the present time, the thymus gland has no known therapeutic effect.

It is very important to remember also that all of these women had anesthesia of some kind during the second stage of labor.

There can be no time limit put on any given case of labor. As long as the uterus is relaxing between pains, the membranes are unruptured, the fetal heart tones are regular and rhythmic, the maternal pulse is under 100, there can be no indication for interference, either medicinal or instrumental, regardless of whether the woman has been in labor eight or twenty four hours.

Dr. Florence W. Austin, Dallas, Tex.—I would like to call your attention to a few things that may occur with patients who have disorders of the internal secretions. We may get results with 2 minims of pituitary solution in a normal individual and we may get no results with two c. c. in hypopituitary patients. We could expect the same variation in results on account of gland dyscrasias in the treatment Dr. Paddock is discussing, as hypopituitary patients occasionally become pregnant in spite of their usual high percentage of sterility.

Dr. Paddock (closing).—I rather expected severe criticism of my paper. From information that I was able to obtain from detail men, one of these preparations alone has in this country at the present time over ten

thousand satisfied users. I have not wished to criticize the manufacturers too severely because they were quite kind in furnishing much of the material used. One of the preparations used was for experimental study and I do not think that it will ever be put on the market.

Dr. Austin's point about the tolerance of the individual patient is one that we must always consider. When we intend to give a patient any pituitary extract, unless we are ready to deliver her immediately under anesthesia, we test her tolerance by giving her the smallest dose possible from which we can expect any appreciable effect. Then if we want to repeat the dose later we can step it up without expecting a violent response.

THE ADVANTAGES OF SPINAL ANESTHESIA*

By CHARLES C. GREEN, M.D.,
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Only a few years ago spinal anesthesia was looked upon with suspicion and was known as a dangerous method used only by eccentric or dramatic surgeons. It was spoken of as a hobby which would soon be relegated to the brush heap along with many other such fads.

Today, however, we have an entirely different attitude. Spinal anesthesia has stood the acid test and is listed among the safest and best methods, and in an unusually large percentage of cases it is the anesthetic of choice.

Its field of practical usefulness has far surpassed the fondest expectations of its most enthusiastic supporters and it is gaining favor with each passing year.

During my recent visit to many of the large clinics in the East, I was most gratified to see spinal used very extensively. In the Post Graduate Hospital, New York, Boston City Hospital, Boston, and Missouri Baptist, St. Louis, spinal anesthesia is in common use.

The time has passed when this form of anesthesia should be used only in those patients whom we expect to die.

Our records show that during the past seven and a half years we have operated under spinal anesthesia upon 835 patients with only one death, which was in a case of severe crushing injury of the leg and thigh. This death is classed as a death from spinal. However, only a few days after this a patient with practically the same injury died in a similar manner while his foot was being amputated under local. This latter case was diagnosed as death from cerebral embolus. In this list of 835 cases are included every character of operation performed below the diaphragm, from gastroenterostomy to amputation of the foot.

There are two methods which I consider satisfactory, but at the Southern Pacific Hospital we stick to the same method which has been in use there for over twenty years, with only some minor changes in the technic. This method is extremely simple. We have an especially small platino-radium needle made for us by Burroughs Wellcome Co., which may bend but will never break. Fifteen minutes before we do our spinal puncture the patient is given an ampoule of ephedrin subcutaneously. We then insert the needle in the first and second lumbar interspace, draw off 1.5 to 2 c. c. of spinal fluid into a small glass, the novocain (1½ to 2½ grains) is dissolved in the spinal fluid and this mixture injected into the spinal canal through the needle which had been left *in situ*. The needle is then withdrawn and the patient allowed to lie down on the table. In about ten or fifteen minutes the anesthesia is established. Prior to the use of ephedrin we had considerable nausea and vomiting as the result of a drop in blood pressure, but we have little or no trouble of this kind now.

The second method is one which we use in cases where we want to do upper abdominal work, and is as follows: make your spinal puncture at the same point and inject a solution of 2 per cent glucose (anhydrous) and 2 per cent novocain in sterile water, using from 1½ to 2½ grains. After the injection the patient is placed in the Trendelenburg position and in this way we secure anesthesia at a higher point than by the first method.

May we caution you about too large doses of novocain? It has been my observation that practically all deaths have occurred where the dose ranged from 3½ to 5 grains, which is entirely too large, unnecessary and extremely dangerous.

With these two methods it is possible to do any abdominal operation, hernia, all classes of gynecological cases, hemorrhoids, amputations of lower extremities, reduction of the lower extremity, dislocations and bone work, with far less postoperative disturbance than with any other method of anesthesia.

Surgeons all over the country are doing every-

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thing possible to get away from ether and narcotics by using gas, local and, as I said before, spinal anesthesia.

In our postoperative check up we have been convinced that there is less nausea, vomiting, less fever and less discomfort from so-called gas pains where spinal is used than following any type of respiratory anesthesia. This results in more rapid convalescence and fewer complications.

Spinal anesthesia makes possible pelvic work in tuberculous patients.

I recall a young woman in the Southern Pacific Hospital whom the medical department was treating for pulmonary tuberculosis. She did well and gained weight during the month, but at each menstruation she had such a severe dysmenorrhea and so much nausea and vomiting as a result of the pain that she lost all she had gained during the month. The surgical department was called upon and we made a diagnosis of pelvic disease and advised operation. Upon opening the abdomen we liberated some clear straw colored fluid and on examination found a bilateral tuberculous salpingitis and a general tuberculous pelvic peritonitis and a fibroid uterus. A supravaginal hysterectomy was performed, both tubes were removed and the peritoneal surfaces exposed to quartz light for one and a half minutes. Following the operation, our patient began to gain steadily and was out of the hospital in ninety days, able to go about her home with practically no rise in temperature. Her condition gradually improved and in eighteen months she was back at work. Here is a patient who could not have been operated upon without spinal and could not have recovered without an operation.

Then in cases of diabetes where any type of abdominal or lower extremity operations must be performed, spinal anesthesia is a life saver. In such cases we have had to amputate legs and operate upon strangulated hernias, so we used spinal and in most cases there was practically no increase in blood sugar. Frequently we have amputated portions of the lower extremities and the patients were able to continue their regular diet without disturbance.

What can be more irritating to an already irritated kidney than ether over a period of thirty minutes to one or two hours? In all such cases spinal anesthesia seems almost a gift from the gods. Also spinal anesthesia gives more thorough and continuous muscle relaxation than any other anesthesia known.

This method of anesthesia is also being widely used by obstetricians and is especially valuable in cesarean sections which become necessary because of some grave kidney lesion accompanied by acute hypertention.

However, its usefulness is not limited to its anesthetic properties. Early in cases of intestinal obstruction it is used to determine the character of the obstruction. If spinal anesthesia is given in early obstruction, the distention will disappear provided no mechanical obstruction exists. Therefore, if the sphincter is relaxed after spinal anesthesia but there is no expulsion of flatus or feces after twenty minutes, you may rest assured that you have a definite obstruction and you may proceed with such surgical interference as may be deemed necessary at this time.

The physiologists also have discovered that spinal anesthesia will materially raise the surface temperature of the lower extremities, and in some of the Eastern hospitals it is being used as a test before sympathectomy is done. In Berger's and other circulatory disturbance of the lower extremities, ganglionectomies or alcoholic injections of the nerve are used in an effort to improve the circulation. But before such work is undertaken it must first be determined whether the destruction of the sympathetics will result in an elevation of surface temperature. If it does, then the work is indicated, but if there is no elevation of surface temperature then the ganglionectomy or the alcoholic injection of the nerve will result in no good. Spinal anesthesia temporarily destroys the sympathetic transmission, and if when you give a spinal there is an elevation of five or more degrees of surface temperature, you have a favorable case for ganglionectomy or nerve injection.

And last, but by no means least, the use of spinal anesthesia improves the surgeon's technic. It does not matter how deep your anesthesia, if you dig and pull on intestines and omentum, your patient will complain and compel you to be more careful and gentle while you are operating.

I cannot conclude this paper without taking cognizance of a recent article by Dr. Gaston Labat. In this article he claims priority for the Trendelenburg position in using spinal anesthesia. He states that he introduced this into the United States in 1920. This is an error, as Dr. R. W. Knox, Retired Chief Surgeon of the Southern Pacific Lines in Texas and Louisiana, was using this position when I first began to practice in 1910, and had been using it for several years prior to this time.

Dr. Labat further states that the use of

ephedrin is dangerous because while the drug does show a stabilization of blood pressure by our method of taking pressure in the extremities, yet after its use there is a lower pressure in the vital centers and a greater load is placed on the splanchnic area.

My answer to this is that physiologically Dr. Labat may be correct and probably he is, because he is well informed, but from the standpoint of practical application he is wrong.

We have a record of over 3,000 cases of spinal anesthesia with two deaths, both of which are doubtfully ascribed to the anesthetic. Of these, over 300 have had ephedrin, not only without a death, but without the extreme drop in blood pressure which was common before its use, and with practically no nausea. Therefore, I feel that the addition of ephedrin adds to the safety of spinal anesthesia rather than increases its danger. This surgical technic I have used three hundred times and from personal observation find that the practical effect does not follow the course of experimental physiology. It matters not to me if all the physiologists disagree with me, I feel safe in continuing its use.

SUMMARY

(1) Spinal anesthesia is on a sound basis today.

(2) It is just as safe as any other type of anesthesia.

(3) It makes possible operation without discomfort in patients who otherwise would be deprived of needed surgical relief.

(4) It is followed by fewer postoperative complications and a more comfortable convalescence than any other type of anesthesia.

(5) It is a great help in determining whether you are dealing with an incomplete paralytic ileus or a definite obstruction.

(6) It is very useful in determining which cases will be benefited by ganglionectomies or alcoholic injections.

(7) It relaxes the abdominal walls more thoroughly and does away with the interference caused by abnormally deep breathing of obese patients.

(8) It improves the surgeon's technic by making him more careful and gentle in his touch.

DISCUSSION (*Abstract*)

Dr. Edward T. Newell, Chattanooga, Tenn.—I am a great believer in spinal anesthesia in selected cases. However, I do not think that it is as safe as any other anesthetic. I want more proof before I will believe that spinal anesthesia is as safe as nitrous oxide and oxygen or ether given by a trained anesthetist; and I am assuming that the spinal anesthesia is given by a surgeon who is fully capable of giving it and who knows how to treat the complications that may arise from the administration of a spinal anesthetic.

Novocain sometimes fails to produce anesthesia. Dr. Green did not say anything about his failures. I should like to ask him whether he has ever entered the canal, injected a spinal anesthetic, everything as far as he knew being correct, the flow normal, and still obtained no anesthesia. Novocain is an uncertain drug. It fails to produce anesthesia in some individuals and it is unexpectedly toxic for others.

It is variously estimated that the mortality in ether and nitrous oxide anesthesia is around one in fifteen to twenty thousand cases. Of course, this is a very indefinite estimation, as many things are to be taken into consideration. Shock and other causes can claim the patient's death.

Spinal anesthesia is the greatest anesthetic in the world for the surgeon, and if we reduce the mortality so that it is as safe as other anesthesias it will be a great boon to humanity. Possibly we may find a drug that is safer than novocain.

Dr. W. W. Harper, Selma, Ala.—Why do we not get a respiratory paralysis when the patient assumes a limited Fowler position? Why does the anesthetic not paralyze respiration?

Can spinal anesthesia be used in cases of shock? For instance, in a man with severe trauma, whose leg must be amputated. What are the limitations of spinal anesthesia?

Dr. John C. Burch, Nashville, Tenn.—The dangers of spinal anesthesia are largely the result of the changes that take place in the circulation. The intact and normal circulation is dependent on three chief factors: these are the efficiency of the heart, the state of the vessels and the volume of blood.

Under spinal anesthesia there is no initial alteration in the heart or the blood volume. The fall in blood pressure is due to a vasomotor paralysis of the peripheral circulatory bed, resulting in an enormous capillary dilatation. This is of no consequence if the venous return to the heart does not drop too much. If it does, the output of the heart becomes decreased and the volume flow of blood to the tissues is decreased. The danger is more potential than real. This is usually of no great consequence. The patient looks and feels as if death were upon him, but he rarely dies. The potential danger lies in the paralysis of the capillary bed, because it is by vasomotor contraction of the peripheral circulatory bed that the body compensates against shock and hemorrhage. If a moderately severe hemorrhage should now take place it would be more serious than in any other condition. It should never be forgotten that spinal anesthesia has the tendency to anesthetize the body's compensating mechanism against the dangers of shock and hemorrhage.

Ephedrin is a good drug and should always be used to prevent or bolster a falling blood pressure. It is not always effective, and when it is not, glucose solutions intravenously should be started. Blood, of course, is

the best of all intravenous fluids in such a circumstance.

Spinal is a good anesthetic. It has its advantages and dangers. It is not now a universal anesthetic. If used cautiously and with regard to its limitations it has a broad and definite field in surgery of the abdomen and lower extremities.

Dr. F. B. Shields, Victoria, Tex.—In our hospital we have had considerable experience with spinal anesthesia, employing spinocain almost exclusively and obtaining very excellent results. The lowering of blood pressure alarmed us at first. Even with the conjoint use of ephedrin, the pressure promptly reached such a level that there was hardly a preceptible difference between the systolic and diastolic reading.

The pulse rate, however, the good color of the patient and the undisturbed respiration gave us confidence and we have continued the use of this form of anesthesia over and above that of ether. Two deaths within six years have marred our records. One was a pregnant woman upon whom a cesarean was about to be performed. The patient suddenly died of a convulsion. The other, an old lady of eighty-five, died almost immediately after the spinal injection.

Dr. Green (closing).—Our technic is very simple. We use a platino-radium needle, 22 gauge, which has been made for us by Burroughs Wellcome Co. This needle has a distinct advantage in that it will not breāk. After inserting it in the spinal canal we collect from 1.5 to 2 c. c. of spinal fluid in a medicine glass. Novocain is then dissolved in the spinal fluid and this solution reinjected into the spinal canal through the needle which is left *in situ* while mixing the drug in the spinal fluid. The amount of drug used depends upon the character of operation to be performed. For amputations and all work below the pelvis we use 1.25 grains of novocain; for pelvic work and also upper abdominal work we use 2 grains, which we consider sufficient for any surgical procedure, including cholecystectomy and gastroenterostomy.

We use an ampoule of ephedrin (¾ gr.) fifteen minutes before we do our spinal puncture, feeling that the ephedrin will reach its maximum effect by the same time that the spinal anesthesia has reached the point where the blood pressure begins to drop. This prevents nausea, and sometimes vomiting, during the early part of our operation. Since the use of ephedrin we have had little or no trouble from lowered blood pressure.

The success of spinal anesthesia in relieving paralytic ileus depends upon early recognition of this condition. If, however, its use is delayed until the intestinal canal has enormously distended so that there is permanent destruction of its nerve supply, then neither spinal anesthesia nor anything else will help.

The use of spinal anesthesia in patients suffering from shock, especially in cases of injury, is extremely dangerous unless something is done to combat the already lowered blood pressure which accompanies shock. We give our patients from 1,000 to 1,500 c. c. of 5 per cent glucose in normal saline intravenously, after which we do not hesitate to use spinal anesthesia and proceed with whatever surgical interference is necessary. In fact, we feel that after spinal anesthesia is well established in these cases, it serves to relieve shock by cutting of all sensation of pain for a period of one and one-half hours or more, from the central nervous system.

We do have failures, averaging, I should say, about one or two a year. The cause for these failures I am unable to explain.

Spinocain was mentioned. I object seriously to the use of this drug, which contains strychnine. The object of spinal anesthesia is to destroy the conductivity of the center fibers leading to the brain. Strychnine does just the opposite, namely: it increases the sensory conductivity of the afferent nerves. Personally I do not think that strychnine is of any use in the treatment of human beings, but is an excellent drug for killing dogs.

Last, the question has been raised of the number of deaths resulting from spinal anesthesia as compared to the number of deaths from respiratory types of anesthesia. My opinion is that the number of deaths at the operating table from spinal anesthesia and respiratory anesthesia is about the same. However, I believe there are numerous deaths caused by pneumonia, suppressed renal function and paralytic ileus following respiratory anesthesia, while following spinal anesthesia such complications are unknown.

TREATMENT OF SOME OF THE COMMONER LID CONDITIONS*

By OSCAR WILKINSON, M.D., D.Sc.,
Washington, D. C.

I beg to present to you my experience in the treatment of the following relatively common lid conditions: blepharitis, chalazion, ectropion, entropion, eczema of the lids, epiphora, nevus and warts, sties, trichiasis and xanthelasma.

From the number of subjects above mentioned you can infer that I am presenting to you a *potpourri.* I realize that a paper could be written on any one of them. My object is to present in a very concise way the methods and remedies that have been most efficacious in my hands in the handling of each condition.

Blepharitis.—This is one of the most common, and at the same time one of the most obstinate, diseases of the lid that we are called upon to treat. We shall discuss it under two headings: squamous and ulcerative blepharitis.

Squamous blepharitis marginalis is the most frequent form, and usually yields to treatment more readily. Its cause must be sought and removed; this is not always easy. This demands the most accurate correction of refractive defects, regulation of the diet, removal of excess carbo-

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hydrates, the giving when indicated of calcium, milk, citrus fruits and cod liver oil, together with sunshine and personal and local hygiene.

The association of the recurrent type of blepharitis with septic tonsils, chronic sinus infections and diseased teeth must never be overlooked. Frequently the removal of these septic foci with mild local remedies will give the most gratifying results.

Medication in squamous blepharitis consists in locally cleansing the margins of the lids with a 5 per cent solution of sodium bicarbonate or some other mild solvent on a cotton swab, after which a thorough massage of the lid borders should be made with a 1 or 2 per cent yellow oxide of mercury ointment. In intractable cases I have found the use of equal parts of citrine ointment and vaseline applied hot to the lid edges, the most effective remedy.

Associated seborrhea of the brow and scalp should be remedied. Where the itching is annoying, a carbolated oxide of zinc ointment is beneficial. Where the lids' margins are thick it is necessary to massage the lids between the thumb nail and a swab of cotton to express the accumulated secretions from the glands of the lids. Any eversion of the puncta, or any lacrymal obstruction must receive attention. For the temporary relief of the disfiguring redness associated with blepharitis I have found an ephedrine ointment effective. The application of hot boric acid fomentations, followed by cold compresses, is beneficial.

Ulcerative Blepharitis.—In ulcerative blepharitis local and general hygiene as indicated above is essential. To remove the crust from the lid margins and to keep it removed is best accomplished by the method recommended by Dr. Chavasse,[1] which is as follows:

> To the lids of both eyes equal parts of ammoniated mercury ointment and vaseline are applied freely. The eyes are padded at night and the following morning the lids are carefully cleansed with a hot 2 to 5 per cent solution of bicarbonate of soda and the ointment reapplied. This treatment is carried out daily. If the crust persists in forming after a few days the cilia are removed. Chavasse says: "Epilation is the best single therapeutic measure available."

I have found the use of citrine ointment and vaseline, equal parts massaged into the lid borders hot once a day, a most effective remedy. Of course the care of the patient's general health and personal and local hygiene is essential. In many cases the proper diet and hygiene are all important.

The use of hot boric acid fomentations with the judicious use of 1 to 2 per cent yellow oxide of mercury ointment will often give the desired relief in connection with the above general remedies. My experience with vaccine has not been encouraging. Recurring pustules of the lid should be carefully touched with 50 per cent solution of carbolic acid or with Lugol's solution of iodine.

Chalazion.—The treatment of this rather frequent tarsal tumor is not always satisfactory. Recurrences are so frequent that at times the symmetry of the lid is seriously damaged.

Treatment of chalazion is local and surgical. The local treatment consists of cleansing of the lid edges, the gentle massaging of them between the thumb nails and the application of 1 per cent yellow oxide of mercury and hot or preferably cold boric acid applications. I have for a long time noticed the association of infected tonsils and adenoids in children, and diseased sinuses and teeth in adults, with recurrent chalazion.

Surgical treatment consists of incising the tumor after it has been thoroughly anesthetized by the use of local massaging of the conjunctival surface with 4 per cent cocaine together with the infiltration of the skin surface with 1 per cent novocaine. I make a vertical incision, and when necessary, a crucial incision, followed by paring of the four corners. The thickened membrane is now thoroughly curetted, and where unduly thick is excised. If not excised, the bottom of the cavity is dried and trichloracetic acid applied, which is thoroughly neutralized with bicarbonate of soda solution.

The lid margin should never be disturbed. Recurrent chalazion is best prevented by attention to the patient's health, correction of the refraction and local hygiene of the lid as above suggested. In badly scarred lids where further scarring is to be avoided, vertical incision with the careful curettage of the chalazion, followed by mild cauterization by means of fulguration, will produce least scarring.

Ectropion.—Ectropion may be classed under three headings: (1) spastic ectropion; (2) senile ectropion; (3) cicatricial ectropion.

Spastic ectropion is due to a spasm of the orbicularis muscle and is usually relieved by the removal of the cause. This is usually seen in young nervous subjects associated with some acute eye disease. The application of a bandage often is sufficient with the correction of other

disturbances present. In obstinate non-inflammatory cases, relief may be had by injection of alcohol between the tarsus and the muscle.

Senile ectropion is frequently seen in elderly subjects who have been suffering from chronic blepharitis, chronic conjunctivitis and obstruction of the canaliculi. It may also be due to atrophy and relaxation of the orbicularis muscle, with or without paralysis, to thickening of the lid margin and distortion of the cartilage. Treatment of the simple senile ectropion consists in the care of associated diseased conditions together with some operative procedure. These are legion, and space does not permit me to discuss them here, but in mild cases I can personally endorse the use of the galvano-cautery as suggested by Ziegler. Where the condition is too extensive to be relieved by cautery, I have found Fox's[2] operation effective.

Cicatricial ectropion requires some operative procedure, and many ingenious operations have been devised. Their number would indicate that none would apply to all cases. Each requires individual study to secure the best results.

Where the lid is still symmetrical, one of the shortening operations is indicated.

In severely scarred cases, due to injury and burns, the best operative procedures require a complete excision of the scar tissue, the complete freeing of the lid border and fixing it to the opposite lid or brow and grafting with the skin of the lid or an epidermal graft. Both these procedures are successful when properly executed.

Entropion.—Entropion may be divided into spasmodic, senilic and cicatricial. Spasmodic entropion is due to a spasm or contraction of the ciliary fibres of the orbicularis and is usually associated with conjunctivitis, corneal ulcerations, too long bandaging of the eye or some inflammatory condition.

Treatment of this condition consists of proper care of associated etiologic conditions, the pulling of lashes away from the eye with adhesive plaster or flexible collodion, and in more severe, especially non-inflammatory cases, the use of the galvano-cautery point as above mentioned, or the injection of alcohol.

Senile entropion is frequently seen in elderly subjects suffering from chronic conjunctivitis and the inturning of the lashes are particularly annoying. This is often associated with a loss of fat from the lid and orbit.

If cold compresses of boric acid and the use of styptic collodion or adhesive do not relieve, the use of the Ziegler cautery point with or without the excision of the strip of skin is always effective.

Cicatricial Entropion.—This is usually due to contraction of the conjunctiva, with distortion of the cartilage from some injury or long continued inflammatory process. The most frequent cause is chronic trachoma.

The treatment of cicatricial entropion is surgical. Here again the number of operative procedures is legion. Moderate entropion of the lower lid can be relieved by excising a narrow strip of skin from beneath the cilia through the entire extent of the lid, after which two rows of cautery punctures are made through the muscle and cartilage after the Ziegler method. The edges of the skin are now brought together with three or four sutures.

In entropion of the upper lid, due to trachoma, the operation of Webster has been most successful in my hands. Webster of Beyrout devised this operation some thirty years ago and has performed it upwards of 5000 times.

The details of the operation are given by Mac Rae.[3] A brief description is as follows:

After a general or local anesthesia, the lid is clamped with a Snellen lid clamp reversed, the flat plate being applied to the skin surface and the lid everted. An incision with a small, narrow knife is made in the conjunctival surface of the lid parallel to the lid margin and about 3 mm. from it. This incision goes from end to end of the lid and right through the tarsal cartilage. It is not desirable to perforate the skin, but it is all essential that the tarsus be completely divided. At each extremity of the lid the incision is curved a little upwards towards the lid margin, but should not extend to the margin. The inner extremity should end just external to the punctum. The lid may have to be shifted in the lid clamp in order to complete the outer ends of the incision. No hair roots should be in the proximal part of the lid.

After the completion of the incision, the clamp is removed and a moist hot pad laid on the lid. A strip of mucous membrane is now excised from the angle of the mouth with a small forceps and curved scissors. This should be about 5 mm. wide and a little longer than the tarsal incision. The strip is laid over the palmar surface of the left hand, raw surface up, from which all fat and submucous tissue is carefully removed with a small curved scissors. Its sides are trimmed parallel and its ends pointed.

The lid is now again everted, the incision cleansed and the strip carefully placed, raw surface down, into the lid wound. The lid is very carefully replaced and pad and bandage applied. In the event of partial failure it is a simple matter to repeat in part or totally without after-disfigurement.

Dr. Wright[4] of Madras says: "I consider that any

surgeon who does not know the operation ought to become familiar with it."

With these remarks I entirely agree, and that, together with the fact that little seems to be known of the operation, is my excuse for describing it at some length.

Eczema of the Lids.—We shall discuss this subject under two headings: acute and chronic eczema.

Acute eczema is both disfiguring and distressing. It frequently appears in persons with delicate skin and is frequently associated with eczema on other parts of the body. There is more redness and swelling about the eye, due to the delicate skin of the lids.

The treatment of acute eczema is both general and local. The general treatment consists of the removal of any apparent etiologic cause, the interdicting of the use of strong soap and water, the use of saline laxatives, the regulation of the diet, the removal of excess of carbohydrates, the use of calcium and cod liver oil.

The local treatment of acute eczema consists in the application of mild astringent remedies, such as mild boric acid, glycerine and camphor lotion. I have found the calamine wash one of the most effective. Where the itching is annoying the use of the ointment of oxide of zinc to which 5 grains of carbolic acid to the ounce are added is very grateful.

In chronic cases with crusting, the ammoniated ointment of mercury is useful. Where there are fissures of the lids, the application of 4 to 5 per cent solution of nitrate of silver is indicated. One of the most satisfactory remedies in my hands is the careful use of equal parts of citrine ointment with vaseline.

In recurrent or intractable cases the x-ray carefully used is very beneficial. The use of a general tonic, especially iron and arsenic, is indicated.

Epiphora.—This very annoying condition is due to excessive secretion of tears or to faulty drainage. Contributing factors are eye strain, emotion, infection of the lids or cornea, misplaced puncta, obstructed or absent puncta, diseased lacrymal sac or duct or obstruction of the nose. Paralysis of the facial nerve is associated with epiphora due to relaxation of the orbicularis.

It can readily be seen that the first step in the treatment of epiphora in a given case is to determine its cause. Any or all of the above conditions must be eliminated or relieved before surgical procedure is to be considered.

Where the punctum is closed it should be gently dilated and the sac irrigated. If the punctum is misplaced, this can often be remedied by the careful use of the cautery point. Where dilatation of the punctum fails I have succeeded by the Basil Graves operation, which I consider very much superior to splitting the canaliculus with the Weber knife. This is done as follows:

The lower punctum is gently dilated and a small knife is slipped into the canaliculus with its edge turned at about 15 degrees angle towards the eyeball. The handle of the knife is now raised and the duct is split about 5 or 6 mm. The conjunctival or bulbar lip of the split duct is now picked up with an iris forceps, and with a rather long pointed Stevens scissors a piece of the inner side of the split duct about 1 mm. wide and 5 mm. long is cut out. A cleansing lotion is used for a week.

I have found this procedure very much more satisfactory than the use of the Weber knife. In infants one can very often open the lacrymal duct by pressing upon the sac with the finger. In a recent case of obstruction of the duct in older subjects, the very careful passage of the lacrymal probe will often give relief.

In chronic dacryocystitis I have been disappointed with probing and the use of styles and the various sac extirpating operations, and now rely on the West operation. With the proper instruments and preparation it is no more difficult than the external sac operations, and it is successful in a high percentage of cases. I feel that it should be mastered by at least one man in each city.

Nevus and Warts.—Small nevi in the lids can be readily removed by fulguration. Larger ones should be removed by the use of radium. Warts and moles can best be removed by fulguration. Excision followed by cauterization with trichloracetic acid is effective.

Sties.—The so-called abortive treatment of sties is usually non-abortive. If seen early, the application of citrine ointment and vaseline applied with a heated probe sometimes inhibits the progress. Cold or hot packs at times give some relief, but early incision is desirable both for relief of the pain and to prevent recurrence. I have found that the best anesthesia can be obtained by painting the line of proposed incision with carbolic acid. The tip of a very fine applicator is covered with a mere strand of cotton twisted tightly, and this is now dipped in carbolic acid and the excess wiped off. The line of incision is now marked off with the carbolized

tip until the skin turns white. After waiting a moment the carbolic acid is neutralized with alcohol. The incision can now be made with very little pain. I have seen no harm from this procedure. In debilitated subjects with recurrent sties, in addition to iron and arsenic and regulation of the diet, I have found the use of stock vaccines often effective. I use increasing doses of *Staphylococcus albus* and *aureus*. Where this does not bring relief, autogenous vaccines are indicated together with a study of the blood sugar.

Trichiasis.—In the treatment of trichiasis one has the choice between electrolysis and surgery.

Where there are but a few hairs present, electrolysis is the method of choice. A very fine needle attached to the negative pole of a galvanic battery is inserted into the hair follicle and a current of 1 or 2 milliamperes is turned on for two or three minutes until bubbles appear at the hair roots. The cilia can now be lifted out with a cilia forceps. Any resistance indicates that the hair follicle has not been sufficiently destroyed. This is both a painful and tedious procedure, and the lid should be injected with 1 per cent novocaine before beginning. A good light and the use of binocular loops are necessary. Where there is extensive involvement the same operative procedure required for entropion is indicated. Here the galvano-cautery is a favorite. Radium carefully used in elderly and sensitive subjects has proved satisfactory.[5]

Xanthelasma.—Here some process of removal is necessary. Trichloracetic acid, full strength, carefully applied to small patches will improve their appearance, but is not always satisfactory.

The use of fulguration is recommended by many and has a place in the partial destruction of very large patches. Excision under novocain anesthesia with sewing up of the wound is still the method of choice.

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DISCUSSION (*Abstract*)

Dr. Jules Dupuy, New Orleans, La.—Blepharitis marginalis is one of the very common lid disorders, which often taxes the patience of both physician and patient to the extreme. It is one of the most difficult conditions to cure. By far the most frequent cause, and the one most often overlooked, is dandruff. I have come to believe that 95 per cent of the cases are caused by this, and until the dandruff is relieved all local treatment is of little avail. Focal infections are responsible in a few cases. I doubt very much that errors of refraction can be considered an etiological factor.

Chalazion is best treated locally by massage with any one of the mild mercurial eye ointments and with moist heat in the form of hot towels. It is the massage and the heat rather than the medication which brings about the absorption. Prolonged treatment of this kind will in many cases avoid surgery. For the past ten years it has been our practice at Charity Hospital to enucleate the cyst *in toto* rather than to use curettage. We seldom have recurrences.

Sties are another very common lid disorder which often tax our patience and ingenuity. I do not agree with the essayist in advocating early incision. It has been my experience that recurrences are more frequent after early incision. If we delay the evacuation of the pus it tends to produce auto-vaccination of the lid. It is almost impossible to anesthetize the part locally, since the injection is often more painful than a quick incision. The teeth and tonsils are very often the source of infection. In children, ethyl chloride as a general anesthetic is ideal for opening sties.

Dr. J. D. Thompson, Port Arthur, Tex.—Blepharitis seems a simple condition to treat, yet we have found it one of the stubbornest with which we have to deal. We may consider the treatment as three-fold: first, removal of the cause or causes, which is not always easy; second, improvement of the general health of the patient; and third, the local application of medicinal agents.

I have been able to cure very few cases of chalazion with medicinal applications, and for this reason I always resort to surgery. When I was an intern in the Sensus Hospital in New Orleans I was taught to incise and curette all chalazions. This treatment was supposed to suffice. But in my private practice, where I had a better opportunity to follow up my cases, I found that a goodly percentage of the larger tumors failed to disappear completely. The very large ones I now completely enucleate, and I get almost 100 per cent cures in cases so treated. In all cases involving the upper lid, whether I am to incise and curette or enucleate, I work from the skin surface of the lid to prevent a possible scar formation on the conjunctival surface which might later prove injurious to the eyeball.

Dealing with the lower lid, if I am to incise and curette, I work from the inside; but if I am to enucleate, I work from the skin surface as in the upper lid. I make my incision horizontally, so that should the operation leave a scar on the skin it will run parallel with the folds of the lids and be less noticeable.

In the treatment of ectropion and entropion, except the spastic type, I use the operation which I think best applicable to the case in question.

The treatment of eczema of the lids is about the same as it would be in any other portion of the body. Treatment of the acute and chronic forms differs. In the acute form, where the lids are red and swollen, we usually resort to one of several solutions that are recommended; but in the chronic type we use one of the various ointments, as diachylon, zinc oxide, white precipitate or ichthyol. The stronger applications are to be avoided.

Epiphora is only a symptom of another trouble. Therefore the treatment must be directed to the removal of the exciting cause. It may be caused by

dachryocystitis, over-functioning of the lacrymal gland, eye strain, irritation from foreign bodies or other injuries or from any condition that interferes with the normal flow of tears into the lacrymal canal and thence into the nose.

I use fulguration in most cases of nevus and warts, either with nitric acid or the electric needle. The larger growths I sometimes remove surgically.

The medicinal treatment of sties in my hands has been very disappointing. They usually suppurate and have to be incised in spite of any treatment. Until they are ready for incision, which I do as early as possible, I use hot fomentations alone, or combined with 1 to 2 per cent yellow oxide of mercury. The hot applications hasten suppuration, afford the patients great relief, and give them something to do while waiting.

Dr. Clifton M. Miller, Richmond, Va.—For the treatment of sties I have been thoroughly satisfied with the results obtained from autogenous vaccines, made by a competent laboratory.

I think we are not dealing with the same trouble in the acute and chronic states of eczema. We must use ointments in the chronic cases and the systemic condition must be considered. In acute cases soothing applications are required, but chronic cases need stimulation.

Dr. J. D. Perdue, Mobile, Ala.—It has been my experience that it is not necessary to enucleate chalazions. In my clinic I see a great number of colored people who are prone to have chalazions and who do not come for relief until they are very large. I curette them very thoroughly and allow the patient to return to work. I feel that the routine enucleation of chalazions is not warranted, especially in the working class of people, since it causes loss of time from their work.

Dr. W. D. Gill, San Antonio, Tex.—In the treatment of acute and recurrent sties I have found the use of bacteriophage dropped into the eye to be a satisfactory treatment. Stock bacteriophage can be used, but it is better if prepared from the patient.

In the treatment of blepharitis we have resorted to autogenous vaccines injected directly into the lid, with very gratifying results in several instances. I think it is a procedure that is well worth a trial in resistant cases.

Dr. Ray K. Daily, Houston, Tex.—I cannot agree with Dr. Wilkinson on the advisability of slitting the lacrymal punctum. I believe that the punctum has a physiologic function in aspirating the lacrymal secretion into the sac, and destroying this function may lead to a permanent epiphora, even when the rest of the lacrymal apparatus is normal. In dealing with epiphora, one must have a clear idea of the underlying pathology. Epiphora in infants is usually due to the persistence of the embryonic membrane at the mouth of the lacrymal duct, which usually disappears spontaneously. This may be assisted by massage over the sac, or by very gentle irrigation. More radical procedures are not justified. In adults, the problem is entirely different. You may have a functional epiphora or an epiphora due to stenosis at some point in the lacrymal sac. In functional epiphora treatment is ineffective. In epiphora due to stenosis treatment should be directed at restoring drainage into the nose. I do not believe in probing the lacrymal duct. By injuring the mucous membrane and causing bleeding, the sac may be left in a worse condition than it was before the probe was passed. Diagnosis may be made by injecting into the sac a few drops of lipiodol and taking a roentgenogram. If the sac shows no dilatation, conservative treatment such as irrigation with astringents, Pregel's iodine, or Besredka's bacterial filtrate may lead to complete recovery. If the roentgenogram shows a dilatation, conservative treatment is a waste of time. Physiologic function can then be restored only by making an opening into the nose above the stenosis, which may be accomplished by the intranasal operation of Halle-West or the operation of Dupuy-Dutemps. We prefer the intranasal operation.

Dr. H. B. Searcy, Tuscaloosa, Ala.—I want to report a method of massage for blepharitis. Most of us have a suction machine, and a suction tube with about a 3 mm. opening in the end. I thoroughly grease the lashes with boric vaseline, and run the suction tube across the lashes several times. It is very easy in this way to evacuate the glands and remove scales. The same suction tube may be used for evacuating sties without the danger of carrying the pus elsewhere. Also in aborting sties we use the same method to clean off the lashes and prevent contamination.

I frequently use a 3 mm. trephine to open a chalazion for curettement.

Dr. Wilkinson (closing).—I did not intend to say that I never use vaccines. Unquestionably we get beneficial results from vaccines in recurrent sties. In the dispensary patients we use stock vaccines, and in our private patients we use autogenous vaccines.

For anesthesia in sty operations, I simply mark the line of incision with carbolic acid, use alcohol, and make the incision. It is a grateful thing to do for your patient. We never inject a lid that is already infected.

PERINAUD'S CONJUNCTIVITIS*

By A. G. WILDE, M.D., F.A.C.S.,
Jackson, Miss.

In reviewing the literature of Perinaud's conjunctivitis, one cannot but be struck with the diversity of opinion regarding its cause, and the variations that have been reported in its pathology and clinical characteristics. While note is usually made of granulations, that is, enlarged lymph follicles of the conjunctiva, at times these are said to be replaced by ulcerations. Looking back upon the history of this clinical conception, it is apparent that more stress has been laid on the coincident signs of early glandular involvement and fever than on the exact picture presented by the eye.

A severe type of conjunctivitis was reported by Pascheff in 1916-1917 that appeared to dif-

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fer from Perinaud's, to which he applied the term *conjunctivitis infectiva necroticans*. Roemer classes Perinaud's conjunctivitis with the recognized types of ocular tuberculosis. Axenfeld also intimated that these were related. Bayer and Herrenschwand found a bacillus infecting rodents that produced a conjunctivitis of the Perinaud type, which they named *Bacillus pseudotuberculosis rodentium*. They also classed the squirrel plague form of conjunctivitis with Perinaud's, akin to the conjunctivitis of Pascheff just mentioned.

Such excellent investigators as Verhoeff and Derby have demonstrated a leptothrix associated with cases of the Perinaud type. Others, studying what they believed to be the same condition, could find no characteristic organism. It therefore seems reasonable to suspect that our clinical conception of Perinaud's conjunctivitis has been based upon a grouping of general symptoms that may arise from various causes, all of which are capable of producing systemic reaction and early glandular involvement.

It is interesting to recall that Perinaud, in describing what he regarded as this clinical entity, laid stress on its association with animals. After the discovery and isolation of the *Bacterium tularense* in 1911-1912 by McCoy and Chapin, Vail identified it in 1914 as the infecting agent in a case of severe conjunctivitis that bore many of the clinical characteristics of the infection that up to that time had been known as Perinaud's. Further study indicates that perhaps the majority of the reported cases of Perinaud's conjunctivitis were due to the *Bacterium tularense*. Evidently many were included from clinical and not laboratory findings. Hence the confusion that we now see in its recognition and classification.

A tabulation of 540 cases of tularemia by Francis showed the oculo-glandular type occurring in 32, or about 6 per cent. Here the primary lesion occurs in the conjunctiva and is usually unilateral. The eye becomes irritated, hyperemic and swollen, and there is a simultaneous involvement of the submaxillary and cervical lymph nodes. Even the parotid has been enlarged in a few instances.

While ulcerations are usually described in tularense infection of the conjunctiva, none was present in the cases here presented, nor did any scars remain after healing. The reaction, namely, malaise, chills, fever and sweats, was simultaneous with the glandular enlargement.

In no instance has it been demonstrated that tularense conjunctivitis has been spread by direct contagion, nor have surgeons been infected while operating upon the involved glands. However, it is one of the most readily transmissible laboratory infections, and many workers have fallen victims after performing autopsies on diseased animals. The incubation time varies from three to nine days.

The diagnosis is made by noting the combination of: (1) history of contact with animals capable of carrying the infection; (2) the development of an ulcer of the skin or severe conjunctivitis; (3) early involvement of the contiguous glands; (4) persistent fever of varying degrees, along with the usual signs of acute illness.

FOLLICULOSIS OF THE CONJUNCTIVA

Confusion in recognizing the many clinical conditions of the eye characterized by an involvement of its lymphatic structures may be clarified by a review of its lymphatic system and a resume of the conditions in which its various portions may become involved.

The lymph structures of the eye are of two general types.

(1) Small lymphoid cell accumulations throughout the palpebral conjunctiva, and perhaps the bulbar, that are normally invisible, but under various types of stimulation can hypertrophy to the stage of follicles. These are circumscribed new formations of typical lymph tissue, and can arise from either chemical or bacterial irritation.

(2) Typical lymph nodes and vessels connected with the orbital region and occurring in four general groupings:

(a) Around the orbital margin of the lower lid and constituting the infra-orbital group.

(b) The facial or buccinator group, lying above that muscle.

(c) The anterior auricular or parotid group, both superficial and deep, and including the well known pre-auricular lymph node.

(d) Lymph drainage can also pass from the lids and accompany the facial veins to the submaxillary group of lymph nodes.

It is thus seen that the lymph drainage from the lids flows sharply away from the mid line, and any or all of these four groups may become involved in inflammatory or malignant conditions of that area. No lymph vessels or nodes

have been demonstrated in the structures of the orbit.

There is considerable reason to believe that small collections of lymph cells exist also in the bulbar conjunctiva. Trantas has just reported (*Arch. d'Ophet.*, vol. 47, July, p. 444) that when a transilluminator is applied to the lower portion of the cornea in incipient cases of trachoma, small perilimbic follicles can be seen at its upper margin with the binocular microscope. He considers these collections of whitish lymphatic tissue specific of that infection.

As an enlargement of the conjunctival lymph follicles occurs in many apparently unrelated conditions, the following list was compiled to learn just where this clinical picture may be expected. This symptom of lymphoid hypertrophy, or better, folliculosis of the conjunctiva, can be encountered in:

(1) The folliculosis of youth, along with general lymphoid hypertrophy of, for example, the tonsils and adenoids. It is very prevalent and usually symptomless.

(2) Follicular conjunctivitis, often indistinguishable from the above, and seldom requiring treatment.

(3) Chemical conjunctivitis, from long irritation usually by eserine, atropine or yellow oxide of mercury.

(4) Inclusion conjunctivitis, or inclusion blenorrhea. In the new born this condition is derived from the genital tract of the mother, and occurs in spite of silver nitrate. It is thus liable to be confused with gonorrheal ophthalmia. In the adult it closely resembles trachoma. These may be two types of infection.

(5) Tuberculosis of the conjunctiva.

(6) Syphilitic infection of the conjunctiva.

(7) Reactions to biologic irritants, for example, ophthalmia nodosa is derived from caterpillar or plant hairs in the conjunctiva.

(8) Sporotrichosis of the conjunctiva.

(9) Blastomycosis of the conjunctiva.

(10) Desquamative epitheliosis of the conjunctiva, caused by cell inclusions, and clinically akin to trachoma, may be the adult type of number 4.

(11) Vernal catarrh, probably a local allergy, and often associated with some form of tuberculous activity.

(12) Conjunctivitis tularense, including the former Perinaud's and Pascheff's conjunctivitis.

(13) The leptothrix conjunctivitis of Verhoeff (pending proof that this is a clinical entity).

(14) Swimming tank conjunctivitis either from infected water or (number 3), a chemical conjunctivitis from agents used in water purification.

(15) Late stages of gonorrheal conjunctivitis.

(16) Trachoma.

It is evident that in addition to the usual folliculosis of the conjunctiva several of the above could be associated with glandular enlargement or even fever, thus producing the general picture of what we have heretofore called Perinaud's conjunctivitis.

When a case presents this symptom of follicular hypertrophy, and the cause is not evident, it is advisable to class it under the general heading folliculosis of the conjunctiva until sufficient time and study have made its nature manifest. It should be recalled, however, that this classification of folliculosis is merely naming a symptom for temporary use, and is no more satisfactory as a final diagnosis than headache or fever.

These lymphoid enlargements were formerly referred to as granulations and trachoma, the most serious affection known to be thus associated, early acquired the name of granulated lids. Since then this term has been taken up so generally as to become a meaningless catch-all for any inflammatory eye condition. As it is thus so indefinite as to convey no real information, it should be entirely dropped.

In an effort to solve the old question of the relationship of follicular conjunctivitis to trachoma, Lindner and Rieger recently cultivated an organism from follicular conjunctivitis that showed the cultural characteristics and form of the *Bacterium granulosis* of Noguchi. Inoculations were made into the conjunctiva of two strains of monkeys, but they produced no results. Lindner then inoculated his own conjunctiva, and a distinct folliculosis developed after three weeks. However, there was no visible inflammation. Two and a half months later the lymphoid hypertrophy had disappeared. It, therefore, seems reasonable to maintain that these two conditions, although showing the common symptom of folliculosis, are distinct, and not different stages of the same infection.

When confronted by a case of possible, but not clearly defined trachoma, one should be wary of arriving at hasty conclusions, there being many similar conditions with which it can be confused. Trachoma is protean in its manifestations, and as we are yet without a definite laboratory technic for its diagnosis, the clinical signs can at times confound the most expert observer.

To diagnose incipient trachoma as a simple form of folliculosis exposes others to the infection and loses valuable time for the victim. On the other hand, to declare a condition to be trachoma when it is not, is to subject the unfortunate patient to much mental anguish, to a prolonged and painful treatment that is not

necessary, and it may economically handicap him in the community.

When one of the simpler forms of folliculosis has been regarded as trachoma, and the lids subjected to rolling or another type of manipulation that produces scar tissue, the end result often defies recognition of the original condition.

When handling the not infrequent cases of this type, it is my custom to divide them into three groups: (1) those that are undoubtedly trachoma and in which rigorous treatment may be started immediately; (2) those that undoubtedly are not trachoma and can with clear conscience be dismissed; (3) the large intermediate group that may or may not develop as trachoma. These are temporarily classed as folliculosis, type undetermined, and are kept under observation until the cause of the lymphoid hypertrophy is found.

It has been my experience that the sudden epidemics of trachoma reported from time to time in the press, especially among children, in localities where it was formerly little known, often belong to this last group, and after mature study are found to be one of the other fifteen conditions in which folliculosis may occur.

Unfortunately, the laboratory is as yet of little use in detecting the early stages of this dread infection. It is to be hoped that by the trans-illumination method of Trantas our diagnosis can be considerably advanced. Otherwise, we have but two tests: (1) that of time, showing in trachoma that the infection persists, and tends to invade the cornea; (2) that of treatment, which causes simple folliculosis to clear quickly and without complications.

CONCLUSIONS

(1) Two cases are reported, each showing sufficient symptoms to be classed as Perinaud's conjunctivitis, although differing considerably in onset, course, and clinical characteristics.

(2) Owing to the confusion regarding Perinaud's conjunctivitis, it is believed that it is probably not a clinical entity, but a general picture that may arise from various causes.

(3) Infection with the *Bacterium tularense* must be considered in severe conjunctivitis of sudden onset, with glandular enlargement and evidences of systemic involvement.

(4) Four distinct groups of lymph structures drain the neighborhood of the orbit, and any or all may become involved in infections or malignancies of that area.

(5) The many known causes of conjunctival follicular hypertrophy may render difficult the recognition of the significance of that symptom.

(6) In incipient and atypical trachoma we have as yet but two diagnostic criteria: the effects of time, and the effects of treatment.

CASE REPORTS

Case 1.—B. B., age 19, complained of inflammation of the right eye and mumps. Her father had died of pneumonia at 54. The mother was living and well at 40. Two sisters, 17 and 21, and one brother, 15, all were living and well. None of the children of the family had died, and no form of eye disorder was known to have occurred in any relatives or associates.

She had had measles, mumps and chicken-pox during early childhood. Although she had never suffered with tonsillitis or other evidences of focal infection, her tonsils were found hypertrophied and removed during 1924. She was born on a farm near Paris, Kentucky, and lived there until 1920, when she came to San Antonio, Texas. She had spent five years in a local orphan asylum, leaving it in 1926. While there, being one of the older girls, she had assisted in the care of the younger children, some of whom had eye affections, the nature of which she did not know. She had recently spent four weeks as a probational nurse in the City Hospital, but could recall no cases of eye infection among those with whom she had come into contact.

Present Illness.—The patient lived in the outskirts of a large military reservation, and one month before she had found a small rabbit in the yard, which she picked up and carried around for about two hours. The rabbit seemed sickly, having a discharge from the nose and eyes. It was later killed and the body destroyed, as it was not deemed suitable for food.

Three weeks later she noted swelling of the right upper lid. Members of her family stated that she became fretful and seemed to have fever, although they did not actually take her temperature. Slight pain then developed in front of the right ear and at the angle of the right jaw. She consulted a physician, who suggested the possibility of mumps, but admitted that the picture was not characteristic of that condition. On account of the swelling of the lid she was referred to the Eye Clinic for examination and treatment.

Physical Examination.—The patient was a white girl in good general health, but slightly anemic. She appeared younger than her age. Her temperature was normal.

Blood examination showed red cells 3,560,000, white cells 9,000, of which polymorphonuclears constituted 70 per cent, small mononuclears 26 per cent, and large mononuclears 4 per cent. Of the polymorphonuclear leucocytes, there were 2 per cent eosinophils and 68 per cent neutrophils. Hemoglobin was 70 per cent.

The left eye was entirely normal, externally and internally. The right upper lid was partially ptosed and distinctly thickened. Its skin was rather dusky in comparison with the other, but no further external changes were visible. There was no interference with lid motility, the apparent ptosis resulting from the swelling.

The color and surface of the conjunctiva of the lower lid were normal. There was no hyperemia, no discharge, no thickening. The transitional fold was not injected. Lying in the outer half of the inferior *cul-de-sac* was a group of enlarged follicles, over which the conjunctiva passed unchanged.

The cornea was normal in surface, brilliance and transparency. The tension was not increased. The anterior chamber and iris were normal. The vision was normal for distance, but a hyperopic correction was worn for near work. The media was clear and fundi were normal.

The upper lid could be everted readily and showed the conjunctiva covering the tarsal surface to be smooth and of normal thickness. The entire retrotarsal conjunctiva was thickened and filled with polypoid masses varying from 0.5 to 2 mm. in diameter. Interspersed through these were small white dots appearing like minute focal necroses. There were no frank ulcerations.

The spaces intervening between the growths were clearly marked, but not of uniform width or depth. The tops of these polypoid growths were rounded and of irregular height. The limbus was not invaded and no skim milk pellicle covered the conjunctiva. It thus differed considerably from the picture of vernal catarrh.

The right pre-auricular lymph node was considerably swollen and slightly painful to the touch. One submaxillary lymph node near the angle of the right jaw, and covering the groove for the facial artery, was enlarged to about 2 cm. in diameter, and also slightly tender. It was firm and clearly outlined, although not freely movable on account of its adherence to the bone. The overlying skin was normal. Viewed from the front, the face was decidedly asymmetric, thus accounting for the suspicion of mumps. There were no glandular enlargements elsewhere in the cervical region, axilla or groins.

Recalling the association with animals that was emphasized by Perinaud, this was first inquired into. It was then that the incident of the sick rabbit was recalled. The patient had never been in contact with either horses or dogs. The family cat was brought to the Clinic and both conjunctivae were found clear. The nictitating membrane was thin and not injected, there was no discharge from either eyes or nose, and its appetite and general activity seemed normal. It was therefore absolved from suspicion as a vector of the infection.

Smears were prepared by scarifying the folliculosis, rubbing it vigorously with a sterile swab and transferring to a slide. Inoculations were made upon media designed especially for the cultivation of the leptothrix. Two of the follicles were removed under aseptic precautions, one being crushed and examined directly for the leptothrix of Verhoeff, the other rubbed over the surface of the medium and then embedded within its substance. These cultures were incubated and watched for several weeks, but all remained sterile. In the crushed specimen, no leptothrix was found after repeated examinations.

As at this time the condition was regarded as purely local, no animal inoculations were attempted, nor were the agglutinating properties of the blood investigated.

Treatment was directed to cleansing solutions and measures designed to increase the hemoglobin. The general health remained good, no fever nor malaise developed, and after three weeks the conjunctival folliculosis returned to normal, with no local scarring. The involved lymph nodes eventually subsided without suppuration.

Case 2.—E. H., colored, aged 52, was referred for treatment on account of an intensely inflamed left eye, September 26, 1930.

His family history was not pertinent. No member had suffered from any eye affection, nor was he aware of any among those with whom he usually associated. He had always enjoyed good health and knew of nothing that could affect his eyes either by infection or trauma.

Five days previously the left eye had become irritable, reddened abruptly, and he developed fever. Almost simultaneously there was seen a swelling in front of the left ear and the following day another appeared under the angle of the left jaw. These were tender on pressure, but not painful otherwise. The following day another lump appeared under the skin at the outer angle of the orbit, which gradually increased in size along with the other two. He was feverish, had considerable headache, was weak, and could not arouse himself to his ordinary duties. Feeling acutely ill, and not knowing the nature of the inflamed eye and these various swellings, he consulted a local physician, who referred him for further examination.

His nutrition was good, although it was evident that he was a sick man. He dropped forward in his chair, was lethargic, opening his eyes slowly, and apparently having some difficulty in comprehending questions and framing an answer. His skin was hot and dry to the touch, temperature 104° F., and pulse 110.

The right eye was normal except for a progressive pterygium at the inner side that was well advanced into the cornea. The lower half of the left conjunctiva was thickened, hyperemic and thrown into deep transverse fold, the depressions between being filled with a thin muco-purulent discharge. There was no ulceration, no follicular enlargement, nor was the membrane unduly tender to the touch. The intense conjunctival injection stopped abruptly at the middle of the cornea. This sharp delimitation was sufficient to suggest its production by some irritating substance that had gotten into the eye. He was positive nothing of the kind had occurred, nor had any form of medication been attempted.

While the picture was then of chemical conjunctivitis, the glandular swelling and fever were more suggestive of Perinaud's infection. But this was rendered doubtful by the violent onset and lack of follicular enlargement. There was no lesion visible that could be classed as a primary focus. The pre-auricular lymph node was considerably swollen, while a submaxillary node at the angle of the lower jaw was greatly enlarged. Attached to the lower outer angle of the orbit, directly below the canthus, was a firm, round cyst-like nodule, 1 cm. in diameter, that was not movable with the skin and tender on pressure.

This last was evidently an involvement of one of the infra-orbital lymph nodes.

On account of his evident general infection he was first given a general examination. The tonsils were hypertrophied and crypts filled. There was an ulcerated lower tricuspid tooth on the side of the involved eye. The urine was negative. The blood showed no

changes beyond 12,600 leucocytes, of which 72 per cent were polymorphonuclears.

A smear scraped from the conjunctiva and stained with Wright's stain showed strands of mucus, pus and desquamated epithelium, with a few staphylococci and short chain streptococci. None was intracellular and in addition they were so scarce that they seemed unlikely to be the invading organism.

Everything considered, the picture was more that of tularemia infection, but smears and growths of that organism are negative and the agglutination reaction would not be present that early. Questions regarding his association with animals revealed that as a farmer he habitually handled horses and mules and was normally surrounded by the assortment of dogs that crystallize around the average negro house and are distinguished more by an abundance of fleas than length of pedigree.

To add a delectable portion to the family larder, as well as to capitalize a plausible excuse to cease labor, he made frequent forays into the adjacent fields in pursuit of rabbits. Usually he was successful and carried his quarry home in a sack or his pocket. He skinned and dressed them himself. Eating the flesh had apparently harmed no one.

Since there is no specific treatment for the condition, the conjunctiva was washed out with boric acid and zinc sulphate solution at frequent intervals, and 1 per cent silver nitrate was applied. Aspirin was given every two hours for relief of pain.

The patient was seen daily, and while his mental sluggishness gradually cleared, his temperature persisted. He was weak and liable to sudden attacks of sweating. Five days later the submaxillary gland became fluctuating, was aspirated, and 10 c. c. of thick yellowish-green pus were withdrawn. This showed no organism either on smear or culture.

Twelve days after the infection started his blood serum was found to agglutinate *Bacterium tularense*. The enlargement of the pre-auricular and infra-orbital lymph nodes continued slowly.

On September 27, or twenty-six days after the onset of the eye symptoms, he became free of fever and except in the swollen lymph nodes, all signs of the infection disappeared. The conjunctiva returned to normal with no scar formation. The pre-auricular lymph node suppurated and was aspirated several times. The infra-orbital lymph node remained tensely swollen with a caseous substance that eventually liquefied slowly and was evacuated.

There was apparently complete recovery. No other glandular enlargements were found. The patient stated that he felt entirely normal and prided himself not a little on the local renown that had been his since the inception of "rabbit disease."

The exact nature of the first case is, of course, open to discussion. The folliculosis was certain, but what lay back of this symptom, common to many conditions, was never discovered. It seemed to be more of the orthodox Perinaud type, but if so, there was no associated leptothrix.

The second case was typically tularense infection, and one of the oculo-glandular types of Francis.

DISCUSSION (*Abstract*)

Dr. J. H. Burleson, San Antonio, Tex.—The two observers who have done most research upon this condition do not agree as to its etiology. Rolandi, after an extensive review of the literature of over 100 cases, is of the opinion that it is of tuberculous origin, but is uncertain whether the infection comes from human or animal exposure. Verhoeff, who has examined many cases, concludes that it is a disease caused by a specific minute filamentous microorganism. This was found in all the cases examined by him and he classified it as a leptothrix.

After a careful review of the literature on this subject my opinion is: first, the disease is a distinct entity of unknown etiology.

Second, it is a local glandular infection which usually lasts about three months; the duration seems to be self-limited, and treatment has no curative value, only palliative.

Third, the presence of tubercle bacilli is only incidental and has no causal relation.

Fourth, neither season nor age seems to affect the disease, though I believe it has been reported more frequently in young persons.

Perinaud, in his first description of this disease, believed that it came from infected animals. This theory has been disproved by later observers. The clinical picture described, which I also have seen in one case, can be confused with only two conditions: trachoma and tuberculous conjunctivitis. Both of these may be ruled out by exclusion. We have none of the sequelae of trachoma, only one eye being affected. There is a clear cornea, little or no photophobia, abundant and loose granulation, the granules being of unusual length, polypoid in appearance, with necrotic tissue at the apex, with involvement of the pre-auricular lymph node and the lymph nodes of the neck. The disease simulates tuberculous conjunctivitis, particularly with the involvement of the glandular system; but when we consider the sudden onset, the character of the granulations, its self-limited course terminating with little or no treatment in perfect recovery, occurring in a healthy individual, we are hardly justified in a diagnosis of tuberculous conjunctivitis.

Dr. Phil M. Lewis, Memphis, Tenn.—Dr. Wilde has not strictly limited his paper to Perinaud's conjunctivitis, but has given us an excellent survey of the various conditions which may be grouped as the folliculoses of the conjunctiva.

Perinaud's conjunctivitis is quite a rare disease. I have not seen a case in over five years which even resembled it. In the summer and early autumn of 1926, I saw two cases which clinically we classified as tularemia, but the agglutination tests were negative and the *Bacillus tularense* was not demonstrated. In Memphis we have two charity eye clinics, whose combined total of new patients averages about 2,000 a year. With the exception of a few cases of tularemia there have been none which could possibly be diagnosed as Perinaud's conjunctivitis.

I agree with Dr. Wilde that it is quite possible that a number of cases reported as Perinaud's were really

tularemia, Pascheff's conjunctivitis, or some kindred disease. The differential diagnosis is certainly not easy, but I should hesitate to state that Perinaud's conjunctivitis was not a clinical entity.

I have always accepted Verhoeff's opinion that the condition was caused by a leptothrix. In 1913, Verhoeff found this minute filamentous organism in 11 out of 12 consecutive cases of Perinaud's conjunctivitis. Since then he has analyzed 18 cases and found the leptothrix in 17 of them. Wherry and Ray, in 1918, reported cultivation of a leptothrix from a case of Perinaud's. Lemoine reported it in one case and Dunphy in two cases. Gifford, in 1927, reported three cases, in two of which he found the Gram positive thread-like organism. In only one of his cases was there a history of close association with animals. Thus several different observers have confirmed the findings of Verhoeff, but a number of others have failed. For example, McKee reported before the American Ophthalmological Society in 1927 a case of Perinaud's, with suppuration of the pre-auricular, submaxillary and supraclavicular lymph nodes, in which careful study of sections stained by Verhoeff's method failed to reveal a leptothrix. He stated that "Frequently fields were seen wherein it was thought leptothrices were present, but closer examination showed them to be degenerated elastic fibers."

Dr. Ray K. Daily, Houston, Tex.—I have some slides of a case of Perinaud's conjunctivitis, in which the test for tularemia was negative. This youngster, 11 years of age, came in from the country with the history of having fever, pain in the right side of his face, and swelling of the neck for 3 three weeks. The right eyelids were swollen and the conjunctiva was studded with fine yellow nodules. The pre-auricular lymph node was still enlarged. The biopsy showed an infiltration with a few polymorphonuclear cells, many lymphocytes, plasma cells, and large monocytes. At one end of the mass was a collection of large monocytes or reticulum cells surrounded by a narrow zone of lymphocytes, some necrosis and very few mitoses.

I do not believe that Perinaud's disease is a distinct entity.

SIGNIFICANCE OF HEMATURIA*

By JOSEPH HUME, M.D.,
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This subject, the significance and importance of blood in the urine, would seem to need no discussion, yet daily experience indicates that the medical public too often fails to appreciate its deadly import. There are still large numbers of patients who have been passing blood for months, very often for years, who have been told that it was due to an acid urine, to the passage of crystals, to ruptured veins, to cold on the bladder, and their natural fears have been soothed with soft words and internal medication. The passage of blood, especially in the absence of pain and in the presence of general well-being, continues to be viewed with nonchalance and indifference by many a practitioner. There are many symptoms and conditions to which people pay no attention, but it is not so with hematuria. In men, the passage of blood is quickly noted and practically always followed by a visit to the physician. In women, some delay is not uncommon, as they confuse it with a disturbance of menstruation. But all these cases seek medical advice early, and any delay in a proper investigation is surely due to the physician. Various compilations show that the time between the first known passage of blood and a proper examination is better than a year, and as new growths account for at least one-half of all hematurias, the seriousness of procrastination can well be understood. It is not difficult, even with a simple outfit, a pair of eyes, a pair of ears, a pair of hands, and a microscope, to get a fair idea of the cause and source of many cases of hematuria. Add to this the cystoscope and the x-ray and the most complicated case can be unraveled.

One should regard a case of hematuria with the feeling that it probably is a serious omen: that such major conditions as tumor, tuberculosis, stone, infection, prostatic disease, nephritis, may be present, and the odds are great that one of these will be found rather than some trivial and insignificant condition. As the history is unrolled, one will note any phenomena antecedent to the onset, the constancy or irregularity of the bleeding, whether it occurs at the start of urination only, at the end only, or is seen throughout the urinary act; whether there is clotting, and if so, whether the clots are long and thin, indicative of a renal origin. The presence or absence of fever, associated with the other symptoms and findings, has its significance. The situation and character of the pain, if present, suggest the anatomical location. In fact, ten questions will give one much important information:

(1) What is the duration of the bleeding?
(2) Is it constant, intermittent or occasional?
(3) Is it slight or profuse?
(4) Is it at the start, at the end, or throughout the urinary act?
(5) Is it painful or painless?

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(6) If painful, what is its location and relation to urination?
(7) Is urination difficult?
(8) Is urination frequent?
(9) Is there fever?
(10) Is there loss of weight?

A few minutes of intelligent questioning will be of much service in arriving at a working diagnosis. Next comes the physical examination, including laboratory tests, all of which are important; but especially, do not forget to palpate the abdomen or to examine the rectum. Too often these two areas are overlooked, and it is one of the ironies of medicine that the very time one omits turns out to be the time that counts. On three occasions I have been able to feel a stone in the kidney by simple abdominal palpation. Next comes a look at the urine, the examination of which may suggest, and at times make, the diagnosis. With women, always use a catheterized specimen, for otherwise the presence of pus, blood and organisms means nothing. With men, use the two or three glass test. With blood in the first glass and the others clear, one sees at a glance that the urethra is the source of the bleeding; with the first and second clear and the third bloody, the source is generally the posterior urethra or bladder; if all are bloody, no definite inferences can be made as to location of the lesion. The presence of pus and organisms, in addition to blood, suggests stone or infection; pus and blood without organisms suggest tuberculosis; blood only, suggests tumor or nephritis. The urine examination coupled with the history and physical examination will allow any practitioner the satisfaction of feeling that he has a fair idea of the nature and location of the trouble, though for exact diagnosis the cystoscope and x-ray are absolutely essential. With the cystoscope, the bladder can be viewed, the ureters catheterized, the separate urines and functions of the kidneys studied, and by the injection of opaque solutions, pictures obtained of the contours of the pelves, ureters and bladder. In the rare cases in which cystoscopy is impossible or inadvisable, intravenous solutions can be given, which, excreted by the kidneys, can be visualized by the x-ray, so that the urinary tract is delineated and accurate deductions as to its condition may be made. By these means it is possible to determine the location of the bleeding in every case of hematuria and to determine the cause in all conditions save one, namely: that called, for the time being, idiopathic or essential hematuria, a renal bleeding, the exact reason for which is at present unknown.

This is only a quick sketch, not a finished picture, and its purpose is to call to your attention certain facts.

(1) The passage of bloody urine means something serious; not 5 per cent of the cases are trivial.

(2) At least one-half of the cases are due to new growth.

(3) The exact diagnosis can and should be made as quickly as possible.

(4) There is a long time, too long a time, between the appearance of the trouble and the institution of proper treatment, and it is the general practitioner who must remedy this situation.

THE MORPHOLOGICAL EFFECTS OF OBSTRUCTIVE JAUNDICE ON THE LIVER OF THE DOG*

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Jaundice[1] is a condition usually associated with certain diseases of the liver and gallbladder. It is characterized by the appearance of bile in urine and blood and discoloration of the skin and sclerae. The more common forms of jaundice are brought about by stagnation of the bile. The disturbance in the outflow of bile leads to its absorption within the liver and the passage of the biliary constituents into the blood and tissues produces the condition of jaundice. For purposes of experimentation jaundice may be induced in animals by ligation and subsequent resection of the common bile duct.

Many researches have been made on the physiological effects of obstructive jaundice in the dog, while little attention has been given to the morphological changes which may result from the condition. Brakefield and Schmidt[1] made a study of the livers of two dogs, while in the human some studies have been made by Schwarz[2] and Eppinger.[3]

Etiologically, jaundice may be classed under two types. The first of these, obstructive or

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hepatogenous jaundice, may be induced in the following ways:

(a) By foreign bodies within the bile ducts, such as gallstones, blood clots or parasites.

(b) By catarrhal swelling of the large or small ducts.

(c) By stricture or obliteration of the ducts, caused by congenital deficiency, or from perihepatitis, from ulcer of the duodenum, or from ulcers or scars in the bile duct.

(d) By obstruction by tumors at the papilla or of the bile ducts, such as carcinoma of the bile duct.

(e) Obstruction by pressure from without, caused by tumors of the liver, tumors of the gallbladder, enlarged glands in the fissure of the liver, tumors of the stomach or duodenum, tumors of the pancreas, a pregnant uterus, or anything which might close the bile duct with pressure from without.

The second type of jaundice is the hematogenous, or toxic type. In this type, the bile is affected by a toxin in the blood and is rendered so viscid that it cannot pass through the bile duct, hence it must back up into the liver and go into the blood stream. Such poisons are:

(a) Definite poisons such as phosphorus, arsenic, snake venom, chloroform, chloral, benzol, and anilin compounds.

(b) Toxins from infectious fevers, such as malaria, pyemia, typhus, typhoid, relapsing fever, scarlet fever, or pneumonia.

Among the many symptoms which characterize jaundice the following may be mentioned:

(a) Many of the tissues are stained yellow.

(b) The conjunctivae show a yellowish tinge immediately after the onset of jaundice.

(c) Bilirubin is found in most of the body secretions.

(d) The urine is highly yellowed.

(e) Skin infections are common.

(f) The stool is clay colored and usually dry.

(g) Often there are serious digestive disturbances and the animal exhibits a general emaciated condition.

(h) The pulse is slow.

(i) Nervous disorders are common.

(j) There is a marked muscular weakness and the animal may go into a state of coma which may be broken by occasional fits of delirium.

Man ordinarily tolerates the condition of jaundice fairly well. The symptoms in man are for the most part comparable to those found in the dog.

It was the purpose of the experiments herein reported to study the histological changes in the livers of dogs in which jaundice had been produced mechanically by ligature and subsequent resection of the common bile duct.

Dogs were used throughout the work. The animals were kept in sanitary cages in a warm room and were observed over a period of two weeks before experiments were begun. The diet consisted of scraps from the cafeteria. No meat was given. Most of the animals ate their food well and maintained a fairly constant weight. Autopsy was made at the end of each experiment to determine whether the dog was normal.

All operations were carried out under morphine-ether anesthesia on healthy dogs weighing about 15 kilograms. An incision was made in the midline. The common bile duct was freed, doubly ligated and about 1 cm. was resected. The median line was closed in the usual manner. The wounds healed promptly, and within forty-eight hours the animals had recovered sufficiently to resume a regular routine of life. Water was supplied at all times.

In preparation of the slides the usual procedures of fixation, embedding, sectioning and staining were employed. All sections were stained with hematoxylin-eosin.

Dog 1, a male, weighed about 15 kilograms.

Sections of normal liver tissue from this animal were studied. The following characteristics identify the tissue of the normal liver of the dog:

(a) The cells have a clear and distinct cell wall and a single nucleus except in occasional cases where two nuclei may be found.

(b) The trabeculae are well defined and the spaces between them are clear.

(c) The bile canaliculi are not confused and their size is constant.

(d) There is but a meagre amount of connective tissue present, and this is found for the most part around the arteries, veins, and bile ducts.

(e) There are no connective tissue cells intermingled with or indiscriminately scattered among the liver cells.

Dog 2, a female, weighed about 15 kilograms.

Sections made seven days after the onset of jaundice show:

(a) The cells were rather distinct.

(b) The canaliculi and the bile capillaries were greatly swollen and filled with bile.

(c) There was little or no increase in the amount of connective tissue.

Dog 3, a female, weighed about 12 kilograms.

Sections made two weeks after the onset of jaundice gave the following picture:

(a) The cells were fairly distinct and in most cases showed only one nucleus.

(b) The canaliculi were gorged with bile and in some cases their walls were ruptured.

(c) There appeared to be a slight increase over the normal amount of connective tissue.

Dog 4, a male, weighed about 12 kilograms.

Sections made three weeks after the onset of jaundice may be described as follows:

(a) The cell walls were partially obliterated and the cells multinucleated. The exact number of nuclei in many cases could not be definitely determined.

(b) The canaliculi were in many cases ruptured, causing a distortion of the trabeculae.

(c) Bile pigments appeared to be diffused throughout the mass.

(d) The amount of connective tissue was considerably increased over the normal and appeared to be spreading from the vessel walls to the intervening spaces.

(e) A number of giant cells were present.

Dog 5, a female, weighed about 15 kilograms.

Sections made six weeks after the onset of jaundice gave the following appearance:

(a) The trabeculae were considerably distorted and the cell walls broken down.

(b) There was a marked increase over the normal amount of interlobular connective tissue.

(c) There was a great number of giant cells.

(d) There were many biliary thrombi around which were broad spaces where formerly blood capillaries were situated.

Dog 6, a female, weighed about 15 kilograms.

Sections made ten weeks after the onset of jaundice had the following appearance:

(a) The cell walls were hardly recognizable and the nuclei were clumped into large masses. On the whole, the liver tissue looked like a mass of nuclei.

(b) Biliary thrombi had increased enormously.

(c) Connective tissue had filled all the intercellular spaces.

Dog 7, a female, weighed about 15 kilograms.

Sections made thirteen weeks after the onset of jaundice gave the following appearance:

(a) Connective tissue had ramified every possible space.

(b) There was an excess number of giant cells.

(c) All structures in the tissue were disrupted.

Studies made on the tissues of the several dogs, as outlined above, show definitely that extensive morphological changes occur in the livers of dogs following the onset of jaundice.

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THE HISTORY OF YELLOW FEVER SINCE THE NEW ORLEANS EPIDEMIC OF 1905*

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The period of yellow fever history which we are considering began in 1905 at the end of the epidemic of that year in New Orleans and extends to the present. When this period opened, the fact had been established by Reed and his associates[1] that yellow fever is transmitted from person to person by the common stegomyia mosquito. This knowledge had been confirmed by the French Commission[2] in Rio de Janeiro and had been most effectively applied to the suppression of yellow fever. Successful control campaigns had been completed or were in progress in Havana, Vera Cruz, the Isthmus of Panama, and Rio de Janeiro.[3]

PREVALENCE AND DISTRIBUTION OF YELLOW FEVER

During the first twenty years of the period, there were many successful campaigns against yellow fever and also much spontaneous disappearance of the disease. The United States was not again invaded, and yellow fever gradually vanished from the West Indies. Successful control campaigns were begun in the Amazon basin —in 1910 at Para,[3] in 1912 at Manaos,[3] and in 1913 in Iquitos.[4]

In 1916 a commission appointed by the Rockefeller Foundation and headed by Gorgas made a survey of yellow fever foci in South America and expressed the opinion that the eradication of yellow fever was feasible.[5] The rapid retreat of the disease gave support to the belief that its elimination from the cities serving as seed-beds of the infection would result in its spontaneous disappearance elsewhere. The complete extinction of the disease was accepted as the ultimate object of yellow fever control.

Further successes against yellow fever were reported. The disease was eradicated from Guayaquil, Ecuador, in 1919,[6] and from Peru in 1921.[7] The whole western seaboard of South America has since then remained free from the infection.

The elimination of yellow fever from Central

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America and Mexico was accomplished less rapidly. There were outbreaks in Guatemala in 1918 and 1920, [8] [9] in Salvador, Nicaragua, and Honduras in 1919,[6] and in British Honduras in 1921.[7] Control measures were instituted and by the end of 1921 it seemed that yellow fever had been banished from these countries. Nevertheless, an unexpected flare-up occurred in 1924 in Salvador,[10] although all Central America had apparently been free from yellow fever since 1921. In Mexico, the disease was unusually active in 1920,[9] involving the eastern and western coasts and the cities of Vera Cruz and Tampico. It was probably continuously present in Mexico in one place or another until 1922, since which date no outbreaks have been recorded.

In 1923 there was a brief epidemic of yellow fever in Bucaramanga in the interior of Colombia.[11] This appearance of the disease came as a surprise, for there had been no recognized yellow fever in the region since the outbreak of 1910 to 1912.

In West Africa yellow fever has been existent for centuries, and it probably originated there.[12] The Yellow Fever Commission appointed by the British Colonial Office in 1913 gathered evidence showing that the disease had long been present and was widely distributed. In 1910 and 1911 it was prevalent over a wide area and there were epidemics in Freetown, Bathurst, Secondee, Accra, and elsewhere.[13] In 1913 there were epidemics in Lagos, Accra, and several smaller places. The alternation of periods of widespread epidemic conditions with intervals of relative quiescence, so characteristic of yellow fever, was a conspicuous feature of the picture in West Africa, where the limited control measures could have had little influence on the general prevalence. It happened to be during one of the lulls that a commission was sent to West Africa in 1920 by the Rockefeller Foundation to study the yellow fever situation. The commission saw no unquestioned cases of yellow fever and was unable to decide definitely whether the African disease diagnosed as yellow fever really was yellow fever, and whether control measures would be feasible.[9]

As a result of the rapid contraction of the yellow fever areas of the New World during the first 20 years of the period we are discussing, a peak of optimism was reached in 1925. It seemed that complete extermination of yellow fever from the Americas and perhaps from the world was practicable and almost in sight. In that year yellow fever was reported from only one region in the Western Hemisphere, northern Brazil, and only three cases were recorded there.[14] Active control measures were being carried on in the principal coastal cities of this region by the Government of Brazil with the cooperation of the Rockefeller Foundation, and it was expected that the area would soon be freed from yellow fever.

In the following year (1926), however, there was a sudden increase in the number of cases in Brazil. The introduction of non-immune troops into the north during political disturbances was probably a factor in the conversion of small unrecognized foci into obvious outbreaks,[15] and in the enlargement of the infected area. In 1928 and 1929 there was a more serious setback. Yellow fever visited Rio de Janeiro in epidemic form for the first time in 20 years. There were 738 cases and 435 deaths[16] before the vigorous antimosquito campaign instituted by the Government brought the epidemic to a close. Small outbreaks are still occurring in neighboring states.

In 1929 there was a small but disconcerting epidemic centering in the town of Socorro in the interior of Colombia.[17] The disease had not been recognized in the district since the outbreak in Bucaramanga six years before, but the introduction of the infection from without seemed highly improbable. Health authorities were convinced that the outbreaks of 1924 in Salvador, of 1923 in Bucaramanga, and of 1929 in Socorro represented the flaring up of infection which had existed unrecognized in the regions for 3, 11, and 6 years, respectively. Moreover, the year 1927 saw an unusual number of outbreaks in West Africa, scattered from Senegal to the Belgian Congo,[16] and in the present year (1931) reports are coming in of numerous appearances of the disease in Gold Coast, Ivory Coast, and the southeastern part of Nigeria.

The study of yellow fever in West Africa was resumed in 1925 by the International Health Board, which created a yellow fever commission under Dr. Henry Beeuwkes for the purpose of the investigation.[14] Since then this commission, with headquarters in Lagos, Nigeria, has been studying outbreaks of yellow fever and carrying on extensive laboratory research. Their observations have made it apparent that it will not be practicable to carry on intensive, countrywide control campaigns until the general sanitary organization in the several countries has progressed much beyond its present state and

until piped water supplies are more generally available in the cities.

The persistence of yellow fever in northern Brazil in spite of extensive control work in the principal coastal cities has caused some apprehension, for a susceptible generation has been growing up in the countries which have been freed from yellow fever and reinvasion might be disastrous. This situation is putting a great responsibility on those in charge of keeping down the stegomyia breeding in seaports and other key cities. While the replacement of the old sailing ships with modern steamships has helped to prevent the spread of yellow fever, the advent of rapid travel by motor-car and by air is threatening to carry the disease into countries which have long been free from it or in which it has never been present.

Although these later years brought a measure of disillusionment to those who had hoped for the early and complete extermination of yellow fever, they brought also a discovery which led to a veritable renaissance in yellow fever research and to the acquisition of new knowledge which is again raising hopes of the ultimate suppression of the disease throughout the world. In 1927, Stokes, Bauer, and Hudson,[18] of the West African Yellow Fever Commission of the International Health Division of the Rockefeller Foundation, found out that rhesus monkeys were susceptible to yellow fever and could be used as experimental animals. A quarter of a century earlier the brilliant investigations of the Army Commission under Reed had made effective control work possible and thus had diminished the urgent need for research in yellow fever, with the result that few persons were engaged in it after the first confirmatory studies. Experimentation on human beings seemed scarcely justified after the method of transmission had been established. The discovery of a suitable laboratory animal at a time when eyes were being opened to the seriousness of the yellow fever situation acted as a stimulus and research was soon going on in Lagos, Accra, Dakar, Rio de Janeiro, Sao Paulo, Bahia, London, Paris, Berlin, Amsterdam, Boston, and New York. The four years since this discovery have added much to our understanding of yellow fever, but have left undisturbed the fundamental facts established by the Army Commission in 1900.

This summary of the world experience with yellow fever during the past quarter-century makes it manifest that:

(1) Yellow fever is firmly entrenched in West Africa and northern Brazil, and is probably endemic in the interior of Colombia.

(2) Cities long free from yellow fever may yet be revisited by disastrous epidemics if the extensive breeding of stegomyia mosquitoes is permitted.

(3) Although it does not seem possible that yellow fever can be eradicated from the whole world in the near future, there is reason to hope that it can be eliminated from the Americas as a preliminary step within a reasonable term of years.

(4) The difficulties in eradicating yellow fever, now more clearly understood, must be met by new knowledge through which the epidemiology of the disease may be studied more effectively and control measures may be strengthened and applied with greater discrimination.

THE SEARCH FOR THE CAUSATIVE AGENT

Prior to 1905, a number of organisms had been announced as the cause of yellow fever, but the claims had been disproved. The Army Commission under Reed[1] had found that the etiological factor could pass through a Berkefeld filter and that it was invisible under the microscope.

In 1909, Seidelin[19] observed certain bodies which he considered to be the parasites of yellow fever, but convincing evidence against this opinion was later presented by the West African Yellow Fever Commission (British).[20]

In 1918, during a yellow fever epidemic in Guayaquil, Noguchi isolated a leptospira from the blood of patients with symptoms indistinguishable from those of yellow fever. The same organism was later obtained by Noguchi and other investigators in yellow fever epidemics in Mexico, Peru, and Brazil.[21] The organism, like the agent studied by Reed and his associates, was filterable through Berkefeld candles. After much experimental work, Noguchi decided that this spirochete was the cause of yellow fever and named it *Leptospira icteroides*. Evidence against this generally accepted view, and in favor of the identity of *L. icteroides* with *L. icterohaemorrhagiae* of Weil's disease, began to accumulate in 1926 and 1927, and has been reviewed elsewhere.[22]

After monkeys had been found to be susceptible to yellow fever, it was possible to make convincing experiments which showed that the etiological agent in yellow fever was not a leptospira and that it belonged to the group classed as filterable viruses.[18] It was shown also by cross-

immunization experiments with monkeys that the yellow fever of Africa is the same disease as the yellow fever of South America,[22] and therefore has the same causative agent.

In 1929, Kuczynski and Hohenadel isolated an organism, *Bacillus hepatodystrophicans,* which they considered to be the etiological agent of yellow fever.[23] They concluded that this organism existed sometimes as a bacillus and sometimes as minute granules. The claims for this organism as the cause of yellow fever have apparently been disproved.[24]

That there are several diseases other than yellow fever which are characterized by jaundice and are confused with yellow fever has become evident. They can be distinguished now from that disease by tests in animals. In all probability the organism which Noguchi isolated in Guayaquil was the pathogenic agent responsible for the illness in certain of the cases presented to him as yellow fever. This leads us to conclude that Weil's disease was present along with yellow fever in Guayaquil in 1918. We have evidence that Weil's disease was present also in the recent epidemic of yellow fever in Rio de Janeiro.[22] Diseases characterized by jaundice have been confused with yellow fever at other times also, for when Guiteras studied the Barbados epidemic of supposed yellow fever in 1916, he decided that the disease was not yellow fever.[25]

While it is now simple to differentiate yellow fever and Weil's disease from each other and from other diseases in general by serological methods, it is not yet possible to identify certain infections other than yellow fever or Weil's disease showing jaundice as a symptom. Such unidentified diseases accompanied by jaundice are being encountered both in Africa[26] and South America. Until they have been named and adequately described as separate entities, there will continue to be confusion.

THE MOSQUITO VECTOR

The role played by the common stegomyia mosquito, *Aedes aegypti,* in transmitting yellow fever has been well known since the work of the Army Commission. Although it still appears that this insect is the principal vector of the disease and perhaps the only important one, it has been shown by recent laboratory experiments that 7 additional African species of mosquito, one other Oriental mosquito, and 3 additional South American species are capable of transmitting yellow fever by bite under laboratory conditions.[27] [28] Including *A. aegypti,* the possible vectors already discovered are 12 in number.

PRESERVATION OF THE VIRUS

When yellow fever was first studied in experimental animals in 1927, it was necessary to pass the virus from animal to animal by transfer of infectious blood or liver tissue. Later the number of animals needed for keeping a strain of the virus going was decreased by introducing mosquitoes into the series. The infection was transferred from monkey to mosquito and again to monkey, and so on. At the present time very few animals are needed for preserving the virus, for we have found that infectious blood or liver tissue, if dried in the frozen state under vacuum and sealed in glass tubes to exclude moisture,[29] may still be infective after two years of storage. By passing the stored strains through monkeys at half-yearly intervals, it is possible to keep a number of strains available for study over an indefinite period.

SUSCEPTIBILITY OF VARIOUS ANIMALS

Several kinds of monkeys, including principally Asiatic and South American species, have been found by a number of investigators to be definitely susceptible to yellow fever in varying degrees. The blood of monkeys which have recovered from experimental yellow fever, like that of convalescent human beings, contains protective antibodies in high concentration. A number of resistant species of warm-blooded animals have been shown to have the power to react to the extent of developing protective antibodies in demonstrable quantities after injections of yellow fever virus.[30] In some animals the injected virus will circulate in the blood for several days after massive intraperitoneal injection, although there is no evidence that infection has taken place.[30] [32]

The outstanding discoveries in the field of animal susceptibility to yellow fever are two. The first, which has already been mentioned, opened the door to the new era of yellow fever research by showing that *Macacus sinicus* and *M. rhesus* are suitable as experimental animals in yellow fever studies. The second discovery, while depending on the first, was of almost equal importance, for it has already greatly facilitated experimental work in the laboratories. In 1930, Theiler,[31] of the Department of Tropical Medicine in the Harvard Medical School, found that white mice were susceptible to yellow fever if inoculated intracerebrally and that the resulting

disease was an encephalitis. With these small and convenient laboratory animals it is now possible to test sera for protective properties against yellow fever and even to titrate for the amount of antibody present.[32] As the virulence of the yellow fever virus for monkeys and presumably for man has fallen greatly with many passages through mice, this adapted virus has opened up new possibilities for protective vaccination. The virus has already been passed successively 175 times through mice and has the constant high virulence and unchanging low incubation period of a fixed virus.

THE PROTECTION TEST

The presence or absence of protective antibodies in specimens of serum from persons or experimental animals may now be determined by performing protection tests in rhesus monkeys or groups of white mice. In these tests both virus and serum are injected in such a way that the animals remain well if there are protective bodies in the serum but die of yellow fever if antibodies are not present. In monkeys the serum is introduced intraperitoneally or subcutaneously and the virus is injected subcutaneously a few hours later. In mice a mixture of the serum and a suspension of virus-containing mouse-brain tissue is injected intraperitoneally and at the same time a small quantity of sterile starch solution is injected into the brain of the anesthetized animal to localize the virus there.[32] Unless the brain of the adult mouse is slightly injured in some such way, the virus circulating in the blood, even in the absence of introduced antibodies, will be unable to attack the brain cells and produce the encephalitis of experimental yellow fever, and the animal will remain well. To titrate sera for antibody content, it is only necessary to perform protection tests in mice, using serial dilutions of the serum to be tested and a constant amount of virus.

The protection test in monkeys was used recently by Beeuwkes, Bauer, and Mahaffy[33] in a study of yellow fever endemicity in Nigeria and Sierra Leone, and by Hudson and Kitchen[34] in 1929 in determining the nature of the then existing outbreak in Socorro, Colombia, and also in confirming the identification of two previous yellow fever epidemics in the neighboring city of Bucaramanga. In epidemiological studies in which mice are being used in protection tests, a much larger number of observations is now being made than would be possible if only monkeys were available, and the results are more easily interpreted.

DURATION OF IMMUNITY

By means of the protection test in monkeys we were able to demonstrate the presence of protective substances in the blood of 5 of 6 persons who had had yellow fever in Norfolk, Virginia, in 1855, 75 years before the blood was drawn, and in the blood of one person who had had the disease in Louisiana 78 years before the specimen was taken.[35] Forty-five of 60 sera from persons in the United States who had had a disease diagnosed as yellow fever from 30 to 78 years previously were found to have protective power. From this study it was concluded that the immunity acquired through an attack of yellow fever ordinarily lasts for life, even when there has been no subsequent exposure to infected mosquitoes.

VACCINATION

The first yellow fever vaccines to be prepared after monkeys came into use as experimental animals were made by Hindle[36] in 1928. By treating liver and spleen tissues of infected monkeys with glycerine and phenol or with formaldehyde, he was able to attenuate the virus to such a degree that it ceased to produce disease in monkeys but nevertheless immunized them. Aragao, Pettit and Stefanopoulo, and Monteiro also produced vaccines by treating infectious monkey tissues with chemical agents. While vaccines of this general type often produced immunity when tested in monkeys, the results in the vaccination of people far from the laboratory were irregular and disappointing,[37] and evidence was secured that the virus in the vaccine rapidly lost its immunizing power with storage.[38] As a result, the use of these vaccines for the protection of persons seems to have been abandoned.

The protection test in mice made it practicable to test the sera of people after vaccination and find out whether they had in fact been protected. This was a large step in advance. It should no longer be possible unknowingly to use an inert substance as a vaccine without finding it out and revaccinating.

The lessening of the virulence of yellow fever virus for monkeys and probably for man during numerous passages through mice made it seem safe to use living virus, with its high antigenic power, in a vaccine. In May, 1931, Sawyer, Kitchen, and Lloyd,[39] after preliminary tests in monkeys, began vaccinating laboratory person-

nel and field workers exposed to yellow fever with simultaneous injections of human immune serum and living yellow fever virus which had undergone over 100 passages in mice. The living virus in mouse-brain tissue was ground up in some of the immune serum. The total amount of serum injected was the same, on the basis of body weight, as the quantity which was found by test to protect rhesus monkeys when given simultaneously with virus strains highly virulent for these animals. The suspension containing the living virus was preserved by drying in the frozen state and storing in sealed tubes. This permitted thorough testing of the material for safety and potency. One lot of the vaccine was used nearly eight months after preparation and found to be effective. Fourteen persons have each been given a single vaccination by this method and all have become immune to the extent of having demonstrable antibodies in their blood. As determined by means of the intraperitoneal protection test in mice, the injection of the immune serum produced no demonstrable passive immunity. An active immunity was established, however, in from 7 to 21 days after vaccination, usually in from 9 to 14 days. The sera of 7 of these persons were tested in monkeys also and protected them against virulent yellow fever virus from monkey source. Titrations of the sera of these vaccinated persons suggest that the antibody content rises for a time after vaccination and is soon about the same as that of persons who have recently undergone attacks of yellow fever.

In presenting the recent history of yellow fever, I have mentioned only a few of the outstanding results of research, placing emphasis on those which seemed of most interest to the epidemiologist and health official. Important studies in the fields of pathology, entomology, and serology have been passed over entirely or barely mentioned. Enough has been presented, however, to give an idea of the fruitfulness of the recent investigations of yellow fever.

THE DANGERS OF YELLOW FEVER RESEARCH

To reveal the new facts about yellow fever many scientists have run serious risks in these last few years of intensive research, and five have succumbed to the disease they were studying: Adrian Stokes, Hideyo Noguchi, William A. Young, Paul A. Lewis, and Theodore B. Hayne. With this illustrious group we must place also the name of A. Maurice Wakeman, who died of an obscure tropical infection contracted while studying yellow fever in West Africa.

Twenty-seven other persons working in yellow fever laboratories have passed through attacks of yellow fever and have recovered.[40] Most of the accidental infections have occurred in persons working with infectious monkey blood or mouse-brain tissue, and it seems that the virus in these cases must have passed through the unbroken skin or mucous membranes in spite of extreme bacteriological precautions.

Now that most of the workers in the posts of special danger have been immunized by accidental infection or through vaccination, there is reason to hope that the series of fatalities has ended. We look back with deep gratitude to the pioneers who laid down their lives in establishing the new era of yellow fever research.

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ACUTE INTESTINAL DISEASES IN INFANTS*

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The purpose of this paper is to discuss the causes of the decline in the death rate of infants under 2 years of age as they have manifested themselves during recent years in the city of Baltimore, and to indicate what may be done further to decrease the mortality in this age group.

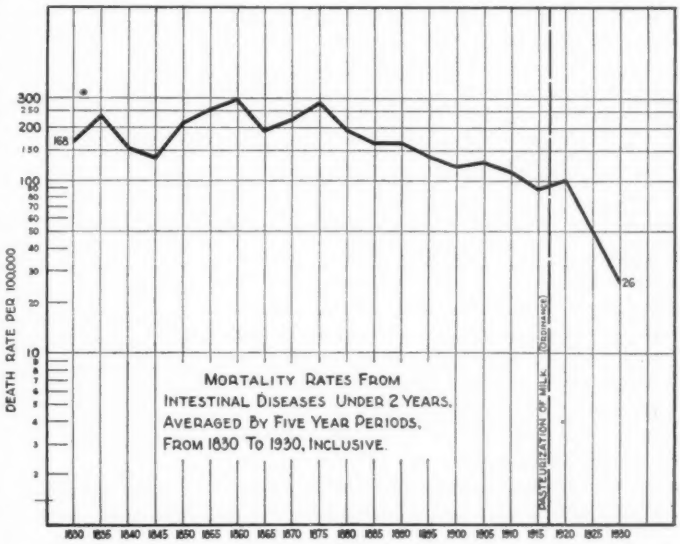
In his "Early History of the Summer Diarrhea of Infants" Booker[1] presented a comprehensive summary of the facts known about the disease up to the beginning of the Twentieth Century. He stated that cholera infantum made its appearance in the towns of the Atlantic coast of the United States about the middle of the Eighteenth Century and extended rapidly into the interior with the opening of that territory. He indicated that the disease had not been recognized in Europe and that it was only after people began to congregate in the urban communities of our own country that it became conspicuous as a separate disease.

Affections of the gastrointestinal tract in infants under 2 years are characterized by a high fatality rate,[2 3 4 5 6] and until very recent years have produced a high mortality in Baltimore. The number of deaths from these diseases recorded under the general heading "cholera infantum" maintained a high level for nearly one hundred years. It is true that the rates show some variations from 1830 to 1875, after which there is a downward trend, but it was not until after 1920 that there was a very marked decline.

As indicated by Howard,[7] it cannot be stated with certainty that cholera infantum as reported and classified in Baltimore up to 1905 was restricted to cases under 2 years of age. Prior to 1899 there were marked variations in the number of deaths under the separate headings "cholera infantum," "teething," and "diarrhea," variations due probably to errors or vagaries of classification rather than to changing incidence of the disease from year to year. Even after combining the deaths reported in the several groups there is a strong possibility that certain others were placed under the heading "dysentery" and not "cholera infantum." It may be safely assumed, then, that the records of high mortality from intestinal diseases ranging in rates from 222 per 100,000 population in 1830 to 139 per 100,000 population in 1905, err probably on the side of being too low (*Chart 1*).

During the period from 1919 to 1930, inclusive, Baltimore has enjoyed a rather sharply declining infant mortality rate. It is significant of the character of child hygiene efforts and extremely pertinent to the discussion of this paper to observe that this decline has been due almost entirely to a decrease in the deaths from intestinal diseases. Indeed, the death rate for infants under one year due to all causes other than intestinal disorders has been practically constant (*Chart 2*).

In general the factors affecting the incidence of acute intestinal diseases in infants have usually been held to be the following: (1) the public milk supply, (2) seasonal temperature, and (3) organized health activities. It is appropriate, therefore, to discuss each of these topics.



MORTALITY RATES FROM INTESTINAL DISEASES UNDER 2 YEARS, AVERAGED BY FIVE YEAR PERIODS, FROM 1830 TO 1930, INCLUSIVE.

Chart 1

*Read in Section on Public Health, Southern Medical Association, Twenty-Fifth Annual Meeting, New Orleans, Louisiana, November 18-20, 1931.

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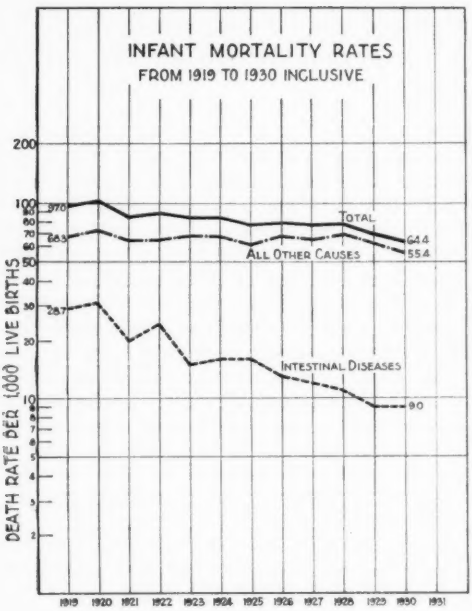

Chart 2

MILK SUPPLY

There is unanimity of opinion on the importance of a high sanitary quality of cow's milk to be used for infant feeding. On the basis of experimental feeding of groups of children with ordinary market milk, Park and Holt,[8] in 1903, observed that a bacterial count of more than 1,000,000 rendered milk unfit as an infant food. The general practice of heating the milk, they said, was certainly a large factor in the lessened infant mortality during the summer months.

Changes in the chemical composition of milk due to bacterial action have a bearing upon infant digestion. The bacterial flora of milk obtained under sanitary conditions consists of four groups: acid coagulating, inert, alkali, and peptonizing. In clean milk the acid group preponderates. Under insanitary conditions of collection and where there is poor refrigeration the alkali and peptonizing groups preponderate. Milk of the latter type contains products of protein disintegration which act as intestinal irritants and produce diarrhea in infants. Ayers,[9] Scholberg and Wallis,[10] and Berard[11] have made valuable contributions to this phase of the subject.

From the point of view of infant feeding a sanitary milk may be defined as one in which the number of bacteria of any character should be low from the time of milking to the time of reaching the gastrointestinal tract of the infant. Both adequate supervision before reaching the home and proper handling in the home are essential.

The first real program in improving the quality of Baltimore milk occurred shortly after the passage of the pasteurization ordinance in 1917. By 1919 the effect of enforcing this ordinance was evidenced by vastly lessened average bacterial counts before and after pasteurization. The bacterial content of the Baltimore milk supply over a period of years is shown in *Chart* 3. The connection between the decline in intestinal disease in infants and the improvement in the sanitary quality of milk as measured by average number of bacteria is apparent.

It is highly significant that at the time during which the milk showed the greatest improvement in sanitary quality there occurred a marked decline in the death rate of infants under 2 years from intestinal diseases. It is indicated that numbers of bacteria in milk alone exert a direct influence on infant deaths.

SEASONAL TEMPERATURE

Since the description of cholera infantum by Benjamin Rush,[12] observers have been struck by the seasonal correlation of temperature with the number of deaths from intestinal diseases in infants. Many have studied this matter with the hope of ascertaining a causal relationship. Brownlee and Young[13] found that epidemics of summer diarrhea are composed mainly of two separate diseases. The first type has its maximum late in July and early in August, and the second during September. They stated further that there is a significant correlation between deaths of the first type and outdoor temperatures in excess of 60° F. but not between the second type and excess temperatures.

Knox[14] observed a group of babies in their homes in Baltimore in 1912 and found that temperature alone was not a potent influence on deaths from intestinal diseases and that babies could be kept well during summer heat of moderate intensity if mothers received proper instruction.

The importance of disturbances in the biological regulating mechanism of the gastrointestinal tract, resulting from maladjustment to external heat, has been emphasized by Arnold.[15 16] He

attributed to heat a depressed gastric secretion which causes a rich bacterial flora not ordinarily present in the duodenum and this in turn brings about nutritional disturbances. Such disturbances, he said, can be prevented and corrected by the feeding of acidified milk.

In 1927, Arnold[17] made a study of the diarrheal death rate in children under 4 years of age in New York City. He observed of recent years a gradual postponement toward the late summer of the usual increase in infant diarrheal deaths. A similar increasing interval between the hot weather season and the time of most infant deaths is found to have occurred in Baltimore during the past two decades. In order to determine the trend of this period of lag of maximum deaths after maximum temperature the day of the year on which the greatest number of fatal cases had their onset was plotted. This is shown in *Chart 4*. A definite trend towards a later date in the year is evident. This phenomenon has an important bearing upon the time of year at which control measures of a public health nature should be concentrated.

Rather than temperature *per se* being the cause of acute intestinal disturbances of infants as was believed by some, it is highly probable that an improved milk supply, better infant feeding and hygienic care have made it possible for young children to suffer less from the heat now than formerly.

PUBLIC HEALTH MEASURES

Diagnosis.—In 1898, Shiga, working on adults in Japan, discovered the *B. dysenteriae* and recognized bacillary dysentery as a distinct disease. This organism was subsequently isolated from adult cases in the Philippines. Later it

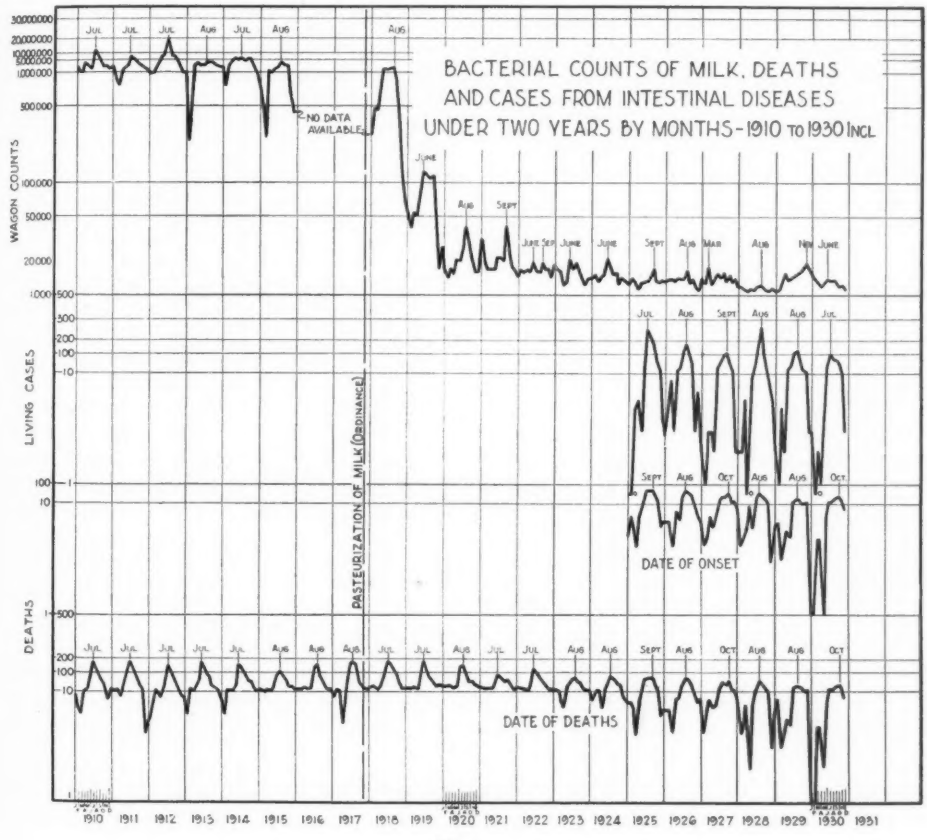
BACTERIAL COUNTS OF MILK, DEATHS AND CASES FROM INTESTINAL DISEASES UNDER TWO YEARS BY MONTHS - 1910 TO 1930 INCL
WAGON COUNTS
LIVING CASES
DEATHS
NO DATA AVAILABLE
PASTEURIZATION OF MILK (ORDINANCE)
DATE OF ONSET
DATE OF DEATHS
1910 1911 1912 1913 1914 1915 1916 1917 1918 1919 1920 1921 1922 1923 1924 1925 1926 1927 1928 1929 1930 1931

Chart 3

was found to be the causative organism in small epidemics in asylums and institutions. Duval and Bassett,[2] working in the laboratory of the Thomas Wilson Sanitarium in 1902, in a series of 42 cases in infants in various forms of the disease, recovered the *B. dysenteriae*. Many workers have gone further and shown that a high percentage of all intestinal diseases in infants, where the predominant symptom is a pronounced diarrhea, have as their cause the dysentery bacillus. Valuable contributions on this subject have been made by Wollstein,[18] Duval and Shorer,[19] Reed,[20] Kendall,[4] and Davison.[5]

Wilkins,[21] in Baltimore, in 1925, followed a group of 628 white infants under 2 years of age, representative of the poorer middle class. In this group he observed that 35 per cent developed intestinal disturbances and of these cases 33 per cent were dysentery.

During the twelve-year period, 1919 through 1930, death certificates of children dying from intestinal disease in the Johns Hopkins Hospital have contained rather complete data regarding diagnosis. Of the total number of 370 deaths thus recorded in infants under 2 years of age, 147, or 40 per cent, were due to dysentery. It will be noted in the following tabulation that the diagnosis was confirmed by autopsy in 70 per cent of the cases.

Table 1

DIAGNOSES OF DEATHS FROM INTESTINAL DISEASES UNDER 2 YEARS IN JOHNS HOPKINS HOSPITAL, 1919-1930, INCLUSIVE.

TESTS CONFIRMING DIAGNOSES	
Dysentery	
Stool culture (positive)	63
Autopsy	64
Stool culture and autopsy	10
Clinical signs	7
Blood (positive)	3
Total	147
Intestinal Disease (Non-Specific)	
Stool culture (negative)	1
Autopsy	187
Stool culture (negative) and autopsy	2
Clinical signs	18
Clinical signs and autopsy	3
Blood culture (negative)	2
Blood culture (negative) and autopsy	1
X-ray	3
Physical examination	5
History	1
Total	223

Davison,[22] from his long clinical and bacteriological experience with dysentery, is of the opinion that in infants a correct differentiation of dysentery from non-specific diarrhea can be made in most instances on the clinical data alone, and where endemic amebic dysentery of children is rare, as it is in the United States, he states that a presumptive diagnosis of bacillary dysentery can be made in those cases where there is a sudden febrile onset and the passage of frequent bloody stools with mucus and pus. From her studies, Reed[20] concludes that mucus and blood in the stools, or simply small quantities of mucus over a long period of time, point to infection with *B. dysenteriae*.

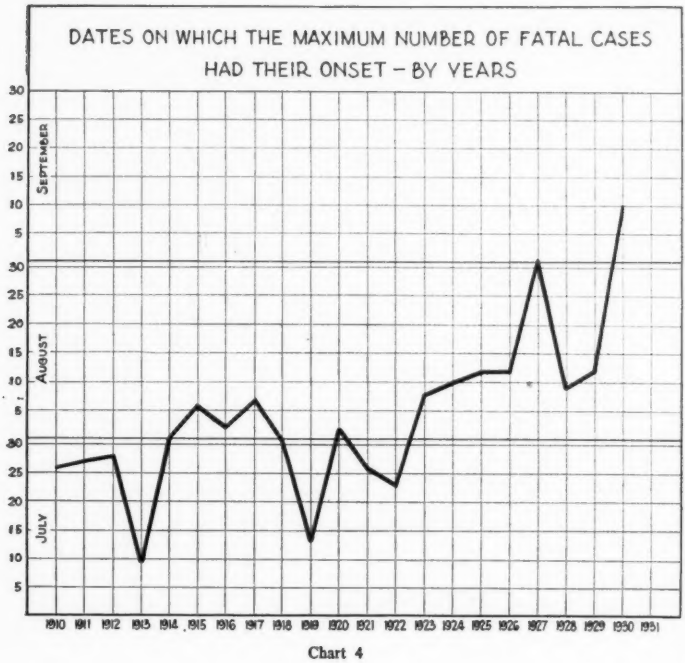

Chart 4

Contact and Carrier Infection. — The importance of contact infection in acute intestinal diseases in infants has been implied or specifically stated by those who have found the *B. dysenteriae* responsible for the infection.[2 18 19 20 4 21 22] Contact infection derived from other children or

adults, within the home as the most potent source, has been emphasized by those who have given careful consideration to this phase of the disease.[23 24] According to Davison,[22] some patients after recovery from dysentery may harbor the bacilli without symptoms for months and even years. He finds that dysentery spreads through the family rapidly and while the older members may have mild diarrheal symptoms the same infection usually produces a severe dysentery in the younger children.

Levy[25] believes that case to case infection is the most important single factor in fatal infantile diarrhea in the South. The proper disposal of all bowel discharges, he said, is the greatest single measure for control of the disease. He attributed the reduction in mortality from 150 per 100,000 in 1911 to 36 in 1919 in Richmond primarily to the educational work of visiting nurses. Peters[23] concluded that while sanitation is important, nevertheless, diarrhea will remain uninfluenced if there are careless habits of living and if cases are not isolated. It was stated by Osler[26] that "isolation of the dysentery patient, with proper disposal of the stools according to the methods used in typhoid fever, should be insisted upon in every case."

Health Education.—The story of health education of mothers of infants in Baltimore dates back to 1884, when the Thomas Wilson Sanitarium, a philanthropic institution, opened a summer home in the country for babies suffering from diarrheal diseases. Booker, and later Knox, were directors of the Sanitarium from 1884 to 1922, and to the pioneer work of the former on the bacteriology and pathology of the intestinal diseases of infants[27] is traceable the first real advance in the control of these diseases in Baltimore. The Sanitarium had provision for 34 mothers with sick babies and 20 infants without mothers. When mothers came with their infants they received educational guidance in the matter of caring for their children, especially as regards the intestinal diseases.

Later, in 1894, the Sanitarium provided a nurse to visit in the homes in Baltimore. She aided in the selection of children to be sent to the institution. During the following three years several part-time nurses were added to the staff. From 1898 to 1903 the Sanitarium employed a nurse throughout the year for home visits with the special object of telling mothers about the Thomas Wilson Service. During the ensuing three years, four and later five full-time nurses were employed on the same basis.

In 1906 the Babies' Milk Fund Association was organized and the Sanitarium nurses were assigned to it. From that time until the present there has been a gradual but steady increase in

Table 2

GROWTH OF HEALTH EDUCATION

Year	Number of Visiting Nurses		Number of Weekly Well Baby Conferences		No. of Special Visits of Instruction by Health Department Nurses
	Thomas Wilson & Babies' Milk Fund Assn.	Health Dept.	Thomas Wilson & Babies' Milk Fund Assn.	Health Dept.	
1894–1897	1 summer months				
1898–1902	1 full-time 1 summer months				
1903–1904	1 full-time 2 summer months		4		
1905–1912	4 full-time		Records not available		
1913–1918	8 full-time		Records not available		
1919	25 full-time	10	Records not available	0	617
1920	22 full-time	18	24	0	1,437
1921	20 full-time	18	24	0	2,146
1922	15 full-time	25	24	1	4,649
1923	25 full-time	25	39	6	5,228
1924	25 full-time	23	41	18	10,091
1925	26 full-time	23	49	20	14,592
1926	30 full-time	23	54	21	7,330
1927	30 full-time	23	55	21	10,482
1928	30 full-time	23	56	21	12,735
1929	30 full-time	23	55	21	11,862
1930	29 full-time	25	48	30	18,945

staff and activities of the combined organizations. The original policy of distributing free milk was soon abandoned and that of instruction of mothers in the home and at well baby clinics was instituted. In 1919, the City Health Department established a Bureau for the Prevention of Infant Mortality, now known as the Bureau of Child Welfare. The Thomas Wilson home was closed in 1922, because by that time the acute, severe cases of intestinal diseases had decreased to such an extent that continuance of the institution was not justifiable.

A brief summary of the growth of these services is given in *Table 2*.

It is noteworthy that the period of sharp decline in deaths from intestinal diseases in infants under 2 years of age is coincident with the years during which the facilities for education along these lines have been greatest.

Knox and Powers[6] showed the value of health education as expressed in public health nursing and well baby conference service. For 1918 they found that the mortality rate was less for that group having nursing supervision than for the city as a whole and still less for the group having both nursing supervision and the conference service of the Babies Milk Fund Association. They stated that with adequate medical and nursing supervision, including instruction in cleanliness and in the proper preparation of milk, diarrheal diseases can be reduced to a minimum.

Health Department Investigation.—On the basis of well defined and easily recognizable symptoms a case history card was drawn up in 1919 for the investigation of cases and deaths from intestinal diseases occurring in infants under 2 years of age in Baltimore. Home visitation and the use of this card by Health Department nurses was begun that year. At this time the case card contained general information regarding the feeding and environmental conditions of the children who had died. After 1924 a more complete history sheet was used for investigation of deaths. This and a similar history card for all living cases reported has been used for the years 1925 to 1930, inclusive.

The history contains three chief items: (1) illness, (2) health and care before illness, and (3) medical attention. The first and second items contain spaces for details regarding the clinical history of the case. From the answers to (1) illness, a fairly accurate conclusion as to the specificity or non-specificity of the disease can be drawn. From the answers to (2) health and care before illness, data can be obtained concerning other illnesses and biological and environmental events in the past history which might have a bearing on the condition at the time of investigation. Each history also carries a question as to any possible source of infection or story of contact. Frequently in the course of her visit the nurse has found that other children in the same family or in neighboring families were suffering with diarrheal disease. In such cases additional histories were made out.

All history sheets have been carefully reviewed with each nurse on her return to the central office. Subsequent study of the case history findings made possible a separation of the cases visited into the following large groups:

(1) *Probable dysentery.* Case histories showing:
  (a) Major symptoms of loss of appetite, fever, vomiting or convulsions.
  (b) Sudden onset.
  (c) Frequent stools (5 or more per 24 hours).
  (d) Consistent pus or mucus (or both) in stools.

(2) *Probable simple or non-specific intestinal disease.* Case histories showing:
  (a) Major symptoms of vomiting or diarrhea or both.
  (b) Gradual onset with no fever.
  (c) Frequent stools (5 or more per 24 hours).
  (d) Consistent watery green stools containing mucus but no pus.
  (e) No blood in stools (drastic purgative excepted).

(3) *Bowel disturbance probably secondary to other pathological processes.* Case histories showing:
  (a) Intestinal disease definitely following other acute illness, most frequently a respiratory disease.

(4) *No bowel disturbance.* Case histories showing:
  (a) Definite information as to the non-existence of any symptoms of bowel disturbance.

These four groups showed the following distribution of deaths and cases:

Table 3

DIAGNOSES FROM CASE HISTORY CARDS

Group	Deaths	Living Cases Reported	Living Cases Found by Nurses	Total	Per Cent
1	692	1145	509	2346	53.9
2	564	837	286	1687	38.8
3	116	13	3	132	3.0
4	173	13	0	186	4.3
Total	1545	2008	798	4351	100.0

Of the deaths due to primary intestinal diseases, 55.1 per cent were classifiable as dysentery and 44.9 per cent as non-specific disease; of the living cases reported and found by nurses 59.6 per cent were classifiable as dysentery and 40.4 per cent as non-specific disease. On the certificates of death during the corresponding period, only 6.6 per cent were recorded as dysentery and 93.4 per cent as non-specific disease; and on the report cards of living cases only 19.0 per cent were specified as dysentery and 81.0 per cent as non-specific disease. From our clinical case history evidence, therefore, it is likely that we are experiencing a greater prevalence of dysentery in both mild and severe cases of intestinal diseases than are indicated from the death certificates or reports. Moreover, the proportion of dysentery to non-specific diarrhea in the cases investigated, is more nearly in accord with the findings of those who have studied the diseases bacteriologically.

Home Visitation.—The plan of having health department nurses make home visits to new born infants, on the receipt of birth certificates, has been followed since the establishment of this work in 1919. Children are revisited at the age of one, two and three years. At the time of her visit each nurse instructs the mother how to prevent the baby from contracting intestinal diseases. Briefly these instructions have included advice (1) to boil all utensils, bottles and nipples used for the baby's food, (2) to wash the mother's hands before preparation of the child's food, (3) to boil all milk and water given, (4) to disinfect and wash the diapers and (5) to screen the house and crib against flies. Demonstrations are given in the home by the nurse whenever this is feasible. The importance of breast feeding is emphasized. The mother is also advised of the necessity for frequent observations of the baby by the family physician. If the family is indigent, it is urged to use the well baby clinic. Great stress is placed on the fact that at the first sign of vomiting or diarrhea, all food given the infant should be stopped at once and the free use of water substituted.

Case Reporting.—Although the diarrhea of infants was placed on report cards as a notifiable disease (diarrhea and enteritis under 2 years of age) in Baltimore in 1922, the number of cases reported was so few as to be comparatively negligible until 1925. In the latter year, the physicians were appealed to by means of circular letters and requested to report all cases of diarrhea of more than five stools in 24 hours. The probability of a bacillary dysentery in the acute cases showing blood and pus in the stools was referred to and they were asked to make this differentiation whenever possible. The response and cooperation of the physicians has been gratifying in the number of cases reported and in the differential diagnosis made on children treated. The latter feature is of special interest in view of the fact that prior to 1925 no cases of dysentery in children under 2 years are on record. Stool containers for diagnostic specimens have been placed at the disposal of the physicians, but few have availed themselves of this service. It is planned, therefore, to make a bacteriological study on cases designated by physicians during the coming year.

The reporting is not entirely complete as many unreported cases continue to be found by our nurses. Nevertheless, it is hoped that the reporting by physicians will reach a high degree of completeness when the invaluable aid to public health education derived therefrom is fully appreciated.

CONCLUSIONS

(1) A rapid decline in deaths from intestinal diseases in infants under 2 years of age has occurred in Baltimore during the past twelve years. During this period there has been an improvement in the milk supply and an increase in health education of the parents concerned.

(2) It is felt that external temperature plays a much less important causative role than was formerly believed, and that environmental adaptability plays a much greater role in diminishing the incidence of these diseases.

(3) The peak of deaths has been showing a tendency to approach the fall season in recent years. This fact will naturally influence the time of year when public health measures will yield the maximum benefit.

(4) Case history cards on deaths since 1924 and on living cases since 1925 show bacillary dysentery as a probable cause in more than half the cases. This proportion had formerly been noted in many bacteriological investigations.

(5) Continued health education with special reference to isolation and proper food stool disposal should be made available if the mortality of these diseases is to be further lowered.

(6) Efforts should be continued along the line of obtaining more complete reporting of cases by physicians, with emphasis on the im-

portance of health education in the private practice of medicine.

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DISCUSSION (*Abstract*)

Dr. Lawrence T. Royster, University, Va.—That infant mortality has declined in recent years is known to everyone and the longevity which is supposed to have been developed throughout the country is due almost exclusively to the elimination of mortality in the first, second and third years of life. Dysentery is definitely infectious. In other diarrheas, certain intestinal influences which Dr. Warthen has named influence the gastric secretions and start the trouble, but that is not all. There are many cases of infectious nature besides the bacillary dysentery cases.

Dr. Warthen spoke of the relationship between temperature and the incidence of this disease. Temperature had an important place in our discussion years ago, but is not so demonstrable today. But since these diseases are largely economic in their origin, they occur almost entirely among people of modest means and one of the things stressed is the keeping of milk in proper condition. There is a relationship between the period of the year and the incidence of these diseases. Poor people, in many instances, have not the means of keeping the milk in good condition. Of course, the pasteurization of milk has reduced the incidence of these diseases. I am a frank advocate of universal pasteurization of milk. My solution is to have but one grade of milk sold on the market and that pasteurized. When we come to that we shall reduce the mortality still further.

A great deal of trouble comes not from the dairy, but from the homes, and people must be educated in the methods of caring for the milk left in their homes. Not until then will we reduce the mortality rate further. It is a matter of education of the lay people, and next and last, but by no means least, there is the general profession. The best education for the lay people is through the visiting nurse, and the more visiting nurses you have in a community the lower the mortality rate is going to be.

Several years ago we made at the University of Virginia Hospital a statistical survey of deaths following diarrhea under two years of age. We felt at that time that dehydration was the first cause of these deaths, and still further that if the dehydration had lasted for as long as an average of twelve days, all the children died. If, on the other hand, the dehydration has lasted for only seven or eight days, the infants had a chance to recover. Beyond that point they did not seem to be able to take care of administered fluids. The lesson then is to educate the general profession to begin the administration of water in prescribed amounts the minute the diarrhea begins and not allow the child to become dehydrated. Another point which I think is worth stressing is that acidified milk has a distinct advantage over any other form of infant feeding in summer time because it has been shown that acidified milk is difficult to infect with pathologic organisms and, therefore, is reasonably safe even when facilities for refrigeration are lacking in the home.

I think that much of the education to the lay public so far as the care and handling of milk is concerned, particularly in the home, must come from milk producers and distributors.

Dr. A. T. McCormack, Louisville, Ky.—The peak of the morbidity and death rate, both from the diarrheas and dysenteries, comes on, approximately, with the conclusion of the holiday season. At the time that a great many people in the cities were going to the country for a few days or weeks, while there and on their way back home, they saw, I imagine, along the roads

a great many indications of sanitary progress being made in the rural sections. Still, there is much to be done.

We must have universal pasteurization of commercial milk and a uniform grade of milk, say grade "A." I was very much interested in driving through Alabama the other day to see the grading of the restaurant and the grading of the milk supply prominently tacked up on the walls of the restaurants in which we took our meals.

Dr. J. Cyril Eby, Plaquemine, La.—Dr. Warthen's data on acute intestinal diseases in infants in Baltimore are just as applicable to rural and urban sections of our Southland.

There was a rapid decline in the mortality rate of infants under two years of age after compulsory pasteurization of milk was adopted in that city. Then, with the establishment of the Bureau of Child Welfare in 1919, a further reduction was noted. This, I am convinced, was due to the education of mothers and to home visits made by nurses to each child.

The great Baltimore fire of 1904 was a blessing in disguise. Since that time millions have been spent on improvements for health protection of that city; improved water supply, sanitary sewerage with modern disposal plants, paving and storm water drains, garbage incinerators, and last but not least, pasteurization of milk.

For over twenty-five years I was a resident of Baltimore and grew up with the modernization of that city. For the past two years I have been doing rural health work in Louisiana. I am glad to state that in a limited way we are able to carry out health and protective measures as to sanitation, improved water and milk supply, and control of communicable diseases. But primarily the education of the public will demand adequate health and protection to its community.

I want to ask Dr. Warthen if sanitary sewerage, for which Baltimore is noted, had anything to do with a decrease in the mortality and morbidity rate in infants?

I agree that proper sterilization of hands, diapers and stools of infants is in order, and also carriers who may transmit acute diarrhea or acute intestinal diseases should be carefully studied.

Dr. Warthen (closing).—In the future we shall further our knowledge of these diseases primarily through arousing the interest of physicians. Case reporting in response to circular letters from the Health Department to physicians has been increasingly good. As a result, cases which might have been neglected are receiving prompt medical and nursing attention.

Dr. McCormack has brought up the question of what might be termed migratory cases. Such cases, when severe, may account for our inability to lower that group of deaths occurring in the fall of the year. City babies, taken to the country for vacation, may develop intestinal disease often in a severe form when there is an absence of immediate attention. This aspect of the control of acute intestinal diseases demands our consideration.

Dr. Eby mentioned the problem of adequate sewage disposal. We have a good and almost complete sewerage system in Baltimore. There is little doubt that it has had a definite influence primarily upon the improvement of general sanitary conditions and secondarily upon the incidence of acute intestinal diseases in infants. However, we must not lose sight of the fact that the method of stool disposal in the infant differs from that in the adult. Therefore, education of mothers along this line will continue to be fruitful.

THE EARLIEST AMERICAN CASUALTIES IN THE WORLD WAR*

By ROBERT U. PATTERSON, M.D., C.M.,†
Washington, D. C.

It is indeed a pleasure to me to be present this evening at this meeting of an association of medical men which represents eighteen states, and which in its influence and accomplishments is second only to that great association which represents the whole nation, the American Medical Association.

As Surgeon General of the Army, I feel that it is a great honor to be privileged to address you this evening. When I was coming down here in the train from Washington, I ran into an old friend, who rather quizzed me to know why it was that I had been selected or called upon to make some remarks on the first evening meeting here in New Orleans. I told him I did not know what the real reason was, but I hoped it was not for the same reason that was applicable to one of our colored soldiers during the War.

There was a negro who lived in one of the Southern states, we might say for purposes of illustration, in Louisiana, who was known as a bad negro. He had been arrested for shooting craps and for being in cutting scrapes, and various misdemeanors in his home town. When the World War came on he was drafted and later went overseas. He became a member of an organization composed largely of colored men from his own town, so that his reputation was well known. His captain was a man who was from the same state and knew all about him. After a few weeks the news was received that Zeke had been made a deacon in the church back home. Of course that caused a great deal of comment and amusement, and his captain, hearing of it, sent for him. He said: "Look here, Zeke, I understand they made you a deacon in

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†Major General, U. S. Army; The Surgeon General.

your church back home. How did that happen?" "Well," he said, "Captain, I dunno jus' how 'tis." The captain said: "You ought to know. You know you are no account, anyway. How could they select you for such a position?" Zeke said: "Cap'n, I dunno, 'ceptin' th' folks back home kinda thought de rough element ob de church oughtta have representation on de board."

In view of the fact that a few years ago I wrote an article which appeared in one of our medical journals on the very subject to which my remarks will be directed this evening, I am not going to read a paper, and will confine myself to talking from a few headings, so that if my memory fails me concerning names, I can refer to them.

On account of the subject of my remarks, this audience is a particularly appropriate one before which to make them, for, finding myself in the great Crescent City of the Southland, I realize that within the confines of the Commonwealth of Louisiana was born, raised, and educated the first man or officer of the Army who was wounded in the World War after we entered into it. That is a circumstance which goes to show that in the late War Louisiana was at the front and foremost. I have always had a strong attachment for the Southland, for I spent more than ten years of my boyhood in your neighboring State of Texas. I have a special feeling of affection for Louisiana because my mother's mother was born in this City 109 years ago. I also have a brother living in your State and a nephew who is attending Tulane University at this time.

As the important address of the evening to which we all are looking forward with pleasant anticipation is to follow my remarks, I am not going into any lengthy discourse, but will be as brief as possible in giving you my message.

It is only right, just, and proper that the facts concerning the order of priority of those who first suffered in the World War in our Army should be made known. If for no other reason, historical accuracy and loyalty to the memory of those who did die and who did suffer first in the World War require a statement of the truth.

As the first casualties in our Army were all members of the Medical Department, there is no audience before whom I could appear that should be better able to appreciate and accept it.

During the War it fell to my lot to find myself in command of United States Army Base Hospital No. 5, which was known locally to the British as the Harvard Unit. That was the second organization to leave the shores of the United States after we entered the War, and it served its entire time with the British Expeditionary Forces in France. I remained with them as commanding officer for nine months, and then was sent from it for other duty in Italy, and later still with our own troops in France. So upon me rests a special obligation to see that the facts are stated. Base Hospital No. 5 was the first organization of the United States Army to suffer casualties in the World War. I repeat, the *first* organization.

Since the World War, there have been a great many statements that have appeared from time to time in the public press which have either been wholly untrue or erroneous through lack of information, many of them no doubt stimulated by a certain amount of local civic pride. And, whenever a statement has appeared in the press and I have seen it, that so-and-so was the first man wounded or the first man killed, I have written to the editor of that paper. Sometimes they wrote me and thanked me for the information; sometimes they did not reply at all. However, I made it my business to get the facts so that there could be no dispute about it should proof be needed.

On the request of the Secretary and Editor of *The Military Surgeon*, which is the organ of the Association of Military Surgeons of the United States, I wrote an article which appeared in that publication in October, 1925. It was called "The Earliest American Casualties in the World War." That is the article to which I have already referred. If anyone is sufficiently interested to wish to check my statements he may read that article, which is in one of the libraries of this City. In it you will find a bibliographical reference for every statement that I make there.

About October, 1927, I happened to be in the City of Washington on business, and while sitting in a theater the movie-tone reproduced the ceremonies of the dedication of a tablet at Governors' Island, New York, to the first soldiers killed in the World War. It was stated that they were members of the 16th Infantry of the First Division; and the Colonel of that regiment was giving the history of the event. I do not want to detract in any way from the honor due these men of that regiment who were killed;

but they were not the first soldiers killed in the World War in our Army. The 16th Infantry was not the first organization to have casualties. On the occasion to which I refer I felt that I ought to do something about it, because here was an official announcement being made by an officer of the Army which was inaccurate. Accordingly I wrote a letter to the Surgeon General of the Army and requested him to take the matter up with the War Department, which he did, with the result that on December 16, 1927, the Secretary of War issued what is known as a press release; that is, he gave out a statement to the press officially announcing the order in which casualties occurred, which should settle the matter for all time.

Before going on to give the names of those who were actually wounded or killed, I think it may be of interest to tell you of some of the earliest military personnel who suffered, not from enemy activity, but who died in one way or another before we actually had any casualties.

The first people who were injured in the World War were injured as a result of an accident and were members of Base Hospital No. 12, which was composed largely of men of Northwestern University in Chicago. This organization sailed from New York on May 20, 1917, and while the crew was having target practice, in order to be prepared to repel submarines, a shell exploded prematurely and a part of the jacket struck a stanchion alongside of the vessel, and fragments from it were deflected and struck three nurses. Two were killed and one was wounded. The killed were Miss Elizabeth Ayres and Miss Mary Wood, and the injured person was Miss Anna Matzen. The ship returned to New York, the bodies were sent ashore, and afterwards the organization proceeded to France. They served within three hundred yards of my own unit at Dannes-Camiers with the British Expeditionary Forces.

The first death that occurred on foreign soil in an organization of the United States Army in France was in the person of a Captain Charles Rund, who was the Quartermaster of my unit (U. S. Army Base Hospital No. 5). Captain Rund was a young man of an unstable nervous type. The responsibilities placed upon him at the outset of the War seemed to be too much for him, and in a fit of depression he took his own life. That occurred on June 6, 1917.

The first death from disease was a young fellow named Colin Campbell, who also belonged to U. S. Army Base Hospital No. 5. He died on June 16 from peritonitis following the sudden perforation of a gastric ulcer.

Now we come to the actual casualties which occurred as a result of enemy action.

The first person wounded in our Army in the World War was Captain Louis Julian Genella, who was born in the State of Louisiana and was brought up and educated in this City. He was wounded in the head and face by shell fire while serving with the British Expeditionary Forces on July 14, 1917. You will recall, some of you, that when the Balfour Mission came to Washington in April, 1917, along with the French Mission headed by Marshal Joffre, the French asked for certain personnel from us immediately. They requested three regiments of railway engineers and a motorized ambulance service. The British asked for base hospitals, three regiments of railway engineers, and as many surgeons and nurses as could be spared. So six Army base hospitals went overseas. As I have already told you, I had command of the second one to leave our country on May 11, 1917. There were a thousand medical officers sent over also, who were loaned to the British. Captain Genella was one of those officers. The second *man* who was wounded in the World War was a *woman,* Miss Beatrice M. McDonald, a nurse, who was a member of Base Hospital No. 2, originally formed from personnel of the Presbyterian Hospital of New York City. She was severely wounded by shell fire on August 17, 1917, while serving at Casualty Clearing Station No. 61, B. E. F. She lost an eye as a result of her wound.

The third person wounded was Lieutenant Philip M. Stimson, Medical Reserve Corps, a cousin of the present Secretary of State, and a brother of the present Superintendent of the Army Nurse Corps. His case illustrates what happens in war when a country is not prepared. There is little time to get ready after a war starts. Here was a skilled pediatrician serving with the forces at the front, but it could not be helped. He had a good basic medical education and he did his duty. He was severely wounded about 2 o'clock in the morning of September 4, 1917, by shell fire while serving with a British Field Ambulance. I saw him the next day when he was brought down to our area, and remember very well the incident.

Now we come to the first people killed.

On the night of September 4, 1917, about 11 o'clock, the organization which I commanded was

stationed at a town called Dannes-Camiers, with the British Expeditionary Forces. It was a bright moonlight night and a group of our officers were standing around talking when we heard a heavy detonation in the distance, and later on another one. We were soon sure that something serious was going to happen. Within a few moments we heard the whirring of an airplane engine overhead, and then the first bombs were dropped. We could hear distinctly the swish of them coming down. Like any sensible man, I called to everyone to "Get down." Well, that order was obeyed with a great deal of alacrity. There were five bombs dropped within a few seconds. First Lieutenant William Thomas Fitzsimons, who was my adjutant, was killed instantly. There were also three privates killed: Privates Tugo, Rubino, and Woods; three lieutenants wounded: Whidden, McGuire, and Smith; and Privates Brown, Mason, Sloan, Stannion, and McLeod were wounded severely. Miss Eva J. Parmalee, one of our nurses, was shell-shocked. She was actually knocked down by the force of one of the explosions. Twenty-two of the British Tommies of the 1,500 we had that night were seriously wounded, and two of them later died as a result of their wounds. As tragic as was that occasion, there is hardly any situation in life that does not have a certain amount of humor. The Harvard Unit was composed of probably as high-powered a group of men as any medical unit that went overseas. I had seventeen specialists out of thirty-five officers, and they were, as Harvard men are expected to be, and all Harvard men are as a general rule, very careful of their diction. When I told these men to lie down when the bombing was about to occur, they did, very promptly, and I joined them. While lying prone I heard somebody breathing hard alongside of me, and I said, "Who is that?" and the officer was so disturbed that he said, "It is me, sir." So it illustrates the mental state of that particular Harvard gentleman on that occasion.

The other thing connected with that occasion that had a humorous side to it, although it was a tragic moment, was the following: Fitzsimons was a very fine young man, what we call in ordinary parlance a "regular fellow." He had not been in the military service long enough to have acquired a knowledge of the usual manner of speaking to a private soldier. He was in his tent when he heard the airplane approaching. Ordinarily a man of say ten years in the service would have said, "Sentry, what appears to be going on here?" But when Fitzsimons stepped to the door of his tent he said to the sentry who was passing on his beat, "Say, Kid, what's doing?" He never found out.

There were some other casualties in our Army very soon after our bombing experience.

The very next day, September 5, 1917, Sergeant Calderwood and Private Brannigan, of the 11th U. S. Engineers, were wounded at Gouzeaucourt. Another medical officer, First Lieutenant George P. Hawes, while serving with the 37th Division of the British Expeditionary Forces, was killed in action on September 28, 1917. There was another young soldier named Ashburn who was wounded on October 29, 1917. He was a member of Company B, 18th U. S. Infantry. And then came the death of the first soldiers who were actually killed in the trenches. They were Corporal Enright and Privates Hay and Gresham, of Company F, 16th U. S. Infantry. These men were killed on the night of November 3, 1917.

What I have given you is the proper chronological order in which casualties occurred in our Army up to November 3, 1917.

After that they became so numerous, especially after the battle of Cantigny in May, 1918, that there is no necessity to go into further detail. However, the fact is that the first seventeen casualties that occurred in our Army were all in the Medical Department.

Lieutenant Fitzsimons was a double graduate of the University of Kansas, having the degrees of A.B. and M.D. After graduation he served as an intern in the German and St. Mary's Hospitals in Kansas City, and later on in the Roosevelt Hospital in New York City.

In 1914, when the World War began, I was serving a detail in the National Headquarters of the American Red Cross in Washington and in September of that year I went over in charge of some hospital units, as we called them, which were sent over to assist all of the belligerents. That is, we took them over and left them there for service. Lieutenant Fitzsimons was a member of one of the two that served in England. Later he served with one of our units at La Panne, Belgium, attached to Dr. De Page's hospital. In 1916 he returned to this country and went back into practice in his home town. He was one of the very first to volunteer for service after our country entered the World War in 1917. He was a fine type of our citizenry, well equipped for his duties, and he met his death in the full tide of his young manhood. He was

the officer who died on September 4, 1917. He was also the first man killed in our Army in the World War because the three privates who were killed at the same time met their end a few minutes after he did.

The United States Army has honored him by naming the largest general hospital we have after him, the Fitzsimons General Hospital, at Denver, Colorado, a hospital with a capacity of 1,800 beds. Tugo Hall is the name of the gymnasium located at Carlisle Barracks, the Medical Department school in which we give our medical officers their field training. It is named for Private Oscar C. Tugo, the first enlisted man killed.

The City of Boston has also named a square in front of the Harvard Medical School after Private Tugo.

In closing I hope you will clearly understand that I am not attempting to minimize the fact that other people died and suffered injuries in the World War. My desire is only to bring out the fact that the Medical Department had the first ones. It was their privilege to be the first to suffer for their country. And that is the message that I have brought to you tonight. It is certainly fitting that an audience of medical men should have that information.

THE TEACHING OF PHARMACOLOGY*

By WM. DEB. MACNIDER, M.D.,†
Chapel Hill, N. C.

The object of presenting this paper before the Section on Medical Education is, first, to trace in a brief fashion the development of this branch of the medical curriculum in order to obtain an appreciation of its value as a science in the curriculum and in therapeutics as both a science and an art. Secondly, from an experience of twenty-seven years in teaching the subject to the average medical student, and from observation of teaching methods in other institutions, I have developed some ideas, which may be correct or incorrect, as to how the subject should be handled pedagogically.

Pharmacology, like the other subjects of the medical curriculum, with perhaps the exception of gross anatomy, has evolved from extensive investigations in an initially ill-defined field of understanding. In a sense, pharmacology is the oldest of the preclinical branches of the medical curriculum, for it primarily concerned itself with an understanding of drugs and other agents which were to be employed in the modification of certain symptoms or more rarely in the eradication of disease. It had its origin when individuals or groups of individuals obtained certain substances and suggested their use in the treatment of disease. From such an unscientific and empirical beginning, pharmacy, materia medica and therapeutics had their origin. For centuries these divisions of medicine in an uninvestigated state held a position in what might be called a medical curriculum. Their place in such a curriculum became open to question as scientific methods of investigation were applied to an understanding of their content, and as time changed the function of the physician for whose use they were primarily intended.

From this jumbled composite of pharmacy, materia medica and therapeutics there emerged first in Germany and England and later in America a highly specialized division of biological information which related itself to certain organic and inorganic materials to which living tissues would react, and this became known as pharmacology. Pharmacology still fails to be a stable evolutionary product. The nature of the subject prevents at present its isolation and stabilization. Its existence depends upon the application of exact physiological, biochemical and physical-chemical methods to its understanding and furtherance. It finds through the employment of such methods a security in the establishment of certain scientific facts which give it its position as an entity in the medical curriculum. In turn, by the use of drugs it has explained especially for the physiologist many problems relative to the functional value of structures and for the biochemist it has elucidated certain problems in metabolism. Pharmacology, however, is unable to stop with the methods of investigation of these sciences. When it is forced to depart from the use of their methods and to apply itself as therapeutics it carries with it as far as it may the science in it, but it is also forced from a lack of understanding to become an art. The element of art in this subject will finally give way to science as research in it, in biochemistry, physiology and pathology provides an exact understanding, first, of the processes of life in nor-

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mal tissues, and second, of the processes in pathological tissues.

Pharmacology is, however, no more unstable as a science than is anatomy, physiology or any of the other premedical subjects. For centuries anatomy rested secure in a simple yet accurate description of anatomical structures. As a science it was superficial. When experimental methods were applied to its understanding, to its beginnings as in embryology, to the factors concerned in tissue growth and differentiation, to regeneration, it became not so certain of its position of exactness. Information upset its complacency. The same type of intellectual shake-up has fortunately happened to physiology, biochemistry and pathology. It is through such readjustments to advances in understanding that science and applied science make progress.

Two processes have arisen in the development of pharmacology which for the present give it a lack of self-satisfaction and self-sustainment. It has evolved from a group of subjects which at one time had but little of the accurate and scientific in them, into a highly specialized division of information which is now as accurate as the knowledge of the physiologist, chemist and biochemist will permit it to be. From the standpoint of a pure science it has isolated itself with certain limitations due to its dependencies. Having obtained through research this degree of security and stabilization, it is asked by the clinician to leave this position of soundness and certainty and make a would-be scientific excursion into the modification and relief of diseased states. The clinician knows very little as a science concerning these states. The pathologist, pathological physiologist and chemical pathologist are just beginning to obtain information through which pharmacology as therapeutics can operate in a less than semi-scientific fashion. Time must again be given to pharmacology to reach its height as an applied science in the treatment of disease. The task which confronts the experimental pathologist is tremendous in its scope and full of adventure both for him and for the pharmacologist.

THE TEACHING OF PHARMACOLOGY

At the outset it becomes difficult to say what is and what is not pharmacology. The subject is vast in its scope and intimately related to other subjects so that its isolation becomes difficult. If medical students must learn it, they do so supposedly to apply it, which raises at once the question not only of the type of course they should receive, but of whether or not the pharmacologist should step over into the domain of the therapist. There are very few courses as such offered in therapeutics. There are fewer departments of this very important subject. The pharmacologists stop with an understanding of the actions of certain drugs on normal tissues. The student wonders why. There is a tremendous gap between the pharmacological laboratory and the clinic.

The question of the scope of pharmacology at once brings forward for consideration not only the number and the character of the drugs which should be included, but also whether or not some slight understanding of closely allied subjects such as materia medica and pharmacy should receive consideration. Any detailed study of these subjects should be eliminated, yet it would appear not amiss for the student to know something pertaining to them, especially in connection with a small number of important drugs such as digitalis, opium and nux vomica. Such information is not primarily useful. Most of the student's education is not immediately useful, but an understanding of the origin and appearance of the important drugs he uses should give to the medical student more respect for himself, if not for the drug. The names of Jenner and Withering make very little difference in so far as a vaccination take or the mechanism of the action of digitalis on the heart is concerned, yet they are good names for the student to know. They cause him to look backward, to find that the past was worth while and that the present fails to tell the whole story.

Pharmacy needs but little space in a course in pharmacology. A short introductory course in this subject to include the technic of prescription writing can do several things. It informs him what to expect of his pharmacist and enables him to have at least some idea as to whether or not his prescription has been faithfully executed. If a physician knows how to write a simple prescription he is more than likely to turn to certain official and dependable drugs to incorporate in this mixture and to turn away from the ever-increasing list of proprietary and incidental mixtures which are forced on him by advertising agencies and drug dispensers. There are certain niceties which the physician may learn from pharmacy in constructing a prescription which is to be introduced into the human organism. A certain combination of drugs may be pleasant to a sick organism. There is an art to medicine.

After excluding as worthless or unnecessary many drugs, there yet remains a fairly large number of very valuable substances both organic and inorganic which should be included in a course in pharmacology. The student should know about them accurately. At the outset it must be understood that I am talking about the teaching of pharmacology to medical students. Such a course may differ and it should differ very much from the type of course which may find its place in a graduate school and be employed to train a person primarily interested in pharmacological research. Such a point of view is, I believe, important. It raises, however, many difficulties in the way of limitations. Just what amount of intricate scientific detail should be included in this course for medical students and how much should be eliminated in the brief time at the disposal of the pharmacologist? It is not in the least to be inferred that a mass of detail is not worth while. It is a question, however, whether or not some of the detail should be sacrificed to an understanding of certain principles which may involve the practical usage of the drugs. As an example, the student should know the composition of Ringer's solution and the principles upon which its conception depends, but is it necessary for him to be so physico-chemically minded that he can explain the delightful intricacies in the action of each constituent?

During the past year I had the privilege of listening to the lecture of a well known pharmacologist on barbital, phenobarbital and certain allied bodies. This entire group of important drugs was dispensed with in less than an hour's lecture. Probably the time limit was adequate. The lecture consisted in a learned and most interesting discussion of the chemical derivation of such bodies with an explanation as to how certain shifts in their structure would influence their action and toxicity. The dose of these bodies was not mentioned and their action on the central nervous system was barely referred to. The lecture should have been given to a group of students interested in pharmaceutical or organic chemistry or to a student in advanced pharmacological research whose interest was in such reactions as this group of drugs would call forth in certain tissues. There was nothing in the lecture for the medical student.

Many teachers of pharmacology have so overestimated and emphasized the purely scientific side of the subject that they have failed to interest the student and have built up in him as a clinician on account of his ignorance a disregard for many drugs of great value.

In presenting a course in pharmacology a question of great interest and importance is that of the method of handling the various groups of drugs. Certain groups, for example, the salts of the heavy metals, can easily be presented from a chemical point of view. They have common chemical characteristics. Even with this rather simple group, if a chemical consideration be largely the sole consideration, the student fails to realize their value as agents in terminating certain important infections. Their specific toxic action may be used to emphasize the value of understanding their chemical structure and the effect of structural changes in lessening their affinity cells of the host and increasing their specific toxicity for certain invading cells.

Certain of the general anesthetic bodies and the group of substances usually classed as hypnotics on account of their similarity in chemical structures may be studied from this point of view. But once they are introduced into tissues, even tissues of the central nervous system, the value of this classification breaks down because it fails to explain their selective action upon different parts of this system, their toxicity for such tissues as blood and their affinity for organs other than the brain and spinal cord.

Certain of the drugs may be grouped and studied on the basis of their remarkable selective affinity for certain groups of cells such as those found in ganglia or for their ability to stimulate or depress various types of sympathetic endings. Such a grouping is of great physiological value and it simplifies the teaching of the pharmacology of the group. It excludes many other important actions and focuses the student's attention upon the effect of these substances on certain isolated structures, and he often fails to grasp the action of the group in its entirety.

We are unable to present the pharmacology of such groups as the circulatory stimulants on a chemical basis or on the basis of the isolated anatomical structures which they influence. Their action on a variety of tissues has to be understood for the entire effect of the substance to be appreciated.

From the foregoing discussion it would appear that no one method of study is entirely satisfactory. I cannot help but feel that the most useful grouping of drugs from the standpoint of the medical student is on the basis of their dominant action on certain systems or groups of

organs, giving adequate attention to special actions which members of the group may have on particular organs, on metabolism and during elimination.

I have the feeling that the laboratory work in pharmacology may be made of particular interest to the medical student and that it furnishes the opportunity to start in him a method of approach to study the reactions of living material to drugs which he should carry into his clinical years and at the bedside so long as he remains interested in the science and art of medicine. Laboratory work may be of value, it may be worthless and it may do actual harm. If the object of the laboratory is to take a guide and by it do and check an experiment, to distill something, mechanically catch the distillate and according to the guide see whether a certain reagent turns it red or blue, then the laboratory becomes not only worthless but harmful. No thought has entered into these mechanical operations. No interest has been quickened by the asking of a question with an attempt to answer it. Such a laboratory is a drab place in its mechanical sloppiness.

In the laboratory which I am thinking about, the student must of necessity have a guide to commence not intricate, but some simple experiments. The guide serves to give him a start and to raise not one but many questions. He introduces something; in this laboratory it is perhaps a drug of known quantity and standard potency into a living, highly complex organism; and then he attempts to explain by observation, references, the aid of his instructors what happened and how it happened. It is of equal importance that he try to understand why this reaction which he anticipated with such certainty failed to happen. As I see it, this point of view is the heart of the pharmacological laboratory. The why and the how are being constantly asked and with a pretty fine enthusiasm the student approaches these riddles which are not mechanical but full of freedom.

A final question which has been previously raised in this discussion may at this point be reconsidered. Regardless of what system has been employed in teaching pharmacology, we may assume that the student has obtained some information concerning what the more important drugs may be expected to do or not to do when they are introduced into living tissue. We wind up our course with a drug and living tissue. Is it sound pedagogy for the work in pharmacology to terminate here? Why has the medical student obtained this information? Has he mastered this division of medical understanding for the sole purpose of the mental training which it imparts and for its biological significance, or has he in addition to this acquired it in order that he may apply it in the modification of symptoms which are harmful, and in the actual cure of disease? I have the belief that he looks forward to using these substances as therapeutic agents. If this be the case, then every department of pharmacology should be so constructed that one member of its teaching force carries the action of the drug a step further and investigates its effects upon pathological animals and at the bedside. When this is done the clinicians will respect pharmacology and be enthusiastic for its advancement, for they will have acquired an understanding of the action of drugs and allied agents not only in normal but in pathological tissues.

Finally, the teaching of pharmacology must depend upon some understanding of the subject and probably the adherence to some general method in its presentation, but after all, as in teaching any subject, the factor of most importance is neither in the method nor the student, but in the attitude, that intangible something of the individual who does the teaching. One teacher describes with scientific accuracy a dead leaf, another teacher with a scientifically adequate description of the leaf transforms it into a living butterfly. It is through such a reach that reality is found.

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DISCUSSION (*Abstract*)

Dr. W. T. Dawson, Galveston, Tex.—It is difficult for the pharmacologist to decide to what he should give detailed consideration in his lectures and laboratory courses. The medical student should learn in the pharmacology laboratory how the actions of drugs may be physiologically analyzed and quantitatively estimated. Some drugs must receive special attention

because of their scientific and some because of their practical importance. The barbiturates have recently come more definitely into the second category, and last year we gave special attention in the laboratory to the dangers of production of general anesthesia by their intravenous administration owing to the extensive clinical experimentation with these agents. The substances emphasized differ from time to time more than the methods for their examination. The student should also learn in a modified chemical laboratory of some sort what he needs to know about the physical and chemical properties of drugs, such knowledge being of importance in various directions, especially in connection with the writing of prescriptions. I feel that the small number of hours necessary to teach the student to write plain and sensible prescriptions should be ungrudingly given by the pharmacology department, since without instruction he is likely to fall into absurd and costly errors.

Dr. Clyde Brooks, New Orleans, La.—I agree with Dr. MacNider as to the value of going back to the history of the important drugs and weaving that into the regular work. In our classes we usually have some special papers prepared by the students on historical subjects, the life of Paracelsus, for instance.

In pharmacology we try to teach the medical student a method that will prove valuable throughout his medical career, if he has not already learned it. I am sure each of you can remember the first time you were assigned a patient and were told "Here is your patient; see what is the matter with him." You had no outside help, and you did your best to solve that case. That stimulated you and interested you. The same method should be used, I think, in pharmacology. We give each student a frog to dissect and examine. The student studies the frog intensively and learns to observe points about the frog which he had not noted before.

At the next laboratory period we give each student a control frog and another frog on which to observe the reactions of an unknown drug. This adds interest to his first work in pharmacology. He learns to rely upon his own observations. We think this is very important, and find it is inspiring to the student to be given an unknown.

I agree with Dr. MacNider that we should not go too deeply into the chemistry and other phases of pharmacology which the student is not able to follow, which should be discussed only in courses in advanced pharmacology. However, we recognize the great value of a surplus of knowledge in pharmacology as in every other subject. No student would be satisfied, if he is the right kind of student, to limit himself merely to the practical knowledge in medicine. We want that surplus of knowledge and want to go as far as we can, yet the course is so short and the time so limited that we must emphasize the points of principal interest.

UNDERGRADUATE MEDICAL TEACHING AND RADIOLOGY*

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Radiology and undergraduate teaching may well be subdivided into two sections: first, teaching by radiology; and second, the teaching of radiology.

TEACHING BY RADIOLOGY

During the past few years, there has been a decided trend toward bringing clinical medicine into closer relationship with the preclinical years. In radiology, we have a definite means of accomplishing this end, for by the proper applications of this modality, we are able to visualize much of the anatomy and pathology of the living and to study numerous physiological processes.

Anatomy.—The usual teaching of anatomy by the examination of the dead tissues, which have lost all of their tone and hence their normal relationship, may well be supplemented by this teaching agency, which enables us to study many of the internal structures in rather minute detail. The relative position of the abdominal viscera may be observed in the living individual by using the various contrast media such as gall-bladder dye for visualization of the gall-bladder, barium for the stomach, and pyelographic contrast media for the urinary tract. The relationship of organ to organ may be accurately demonstrated, showing the innumerable variations which must be considered within the limits of normal. The true height of the diaphragm may be shown. A person who has never seen the diaphragm under the fluoroscope, or on an x-ray film, has never seen it as it really exists in the living normal individual. At the autopsy or dissecting table, the nerve tone is of course absent, and the diaphragm has risen to the level seen after section of the phrenic nerve. The arrangement of the bronchial tree may be beautifully demonstrated by stereoscopic films of the chest after the injection of iodized oil. The osseous structure, of course, may be studied in much greater detail by the use of x-ray films in conjunction with the study of the skeleton,

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as not only the external markings are seen, but the inner structure is also revealed. Much of the muscular structure, as well as the skin and subcutaneous tissues, may now be shown by carefully made films. The subarachnoid spaces in the cranium with the ventricular system and the spinal canal can all be demonstrated by air injection, which is in rather common use in encephalography.

The foregoing are only a few of the ways in which radiology may aid in teaching living human anatomy. Aside from more accurate demonstration, it would seem that such teaching would certainly tend to make the study of anatomy more interesting, for the radiological studies are on living persons, whereas the usual preclinical anatomy is only a study of the dissecting room subject, which is, looks, and smells dead.

Physiology.—Physiologists are much indebted to radiology for the demonstration of many physiological phenomena in the body, and yet it is surprising how relatively few teachers use radiology in teaching. Practically the entire alimentary canal and its movements may be studied. Diaphragmatic and cardiac movements may be seen, and yet how few of our medical schools have a fluoroscope in their physiological laboratories! The peristaltic action of the ureters and bronchioles has been beautifully shown by serial roentgenograms, but this is seldom, if ever, demonstrated to medical students. The study of gall-bladder function has received much attention during the last few years, and it may well be studied, using the gall-bladder dye as both a visualizing agent and as a test of function.

This does not by any means exhaust the teaching possibilities of radiology as applied to physiology.

Pathology.—In the teaching of pathology, the usual custom is to study the gross autopsy appearance and the histological structure. Why not at the same time give also the x-ray appearance of the pathological changes? The roentgen ray is, in a manner of speaking, a gross microscope, which enables us to differentiate varying tissue densities; and by study of the arrangement of the densities, in the majority of cases, we are enabled to make a correct tissue diagnosis in the living. This being the case, it would seem that the radiological appearance should form an integral part of any course in pathology.

In the foregoing discussion of teaching by radiology, there is nothing new presented, but it is felt that such teaching should be more generally used. In several of our medical schools, methods similar to those described have been in use for several years, but such courses are the exception rather than the rule.

TEACHING OF RADIOLOGY

In the undergraduate teaching of radiology, as in the teaching of other specialties in the undergraduate years, it is well to remember that we are not training specialists during the period of the four-year medical course. The scope of such a course should embrace four cardinal points: first, a very brief history of the discovery of the roentgen ray, with subsequent developments; second, a very brief description of the physics of the apparatus; third, a rather full outline of the uses and limitations as regards diagnosis and treatment; and fourth, just sufficient detail as to technical interpretation and thereapy, for the student properly to evaluate radiology as a possible life work.

The story of the discovery of the roentgen ray is one of the most interesting portions of scientific history, illustrating, as it does, a true scientist's zeal in running to earth an unusual phenomenon encountered in a physical laboratory. Roentgen was a scientist of the highest order, and his work and discovery should be an incentive to any student to do more thorough work. Since the discovery of the x- or roentgen rays, steady progress has been made in the facility with which these rays are used. As in so many other branches of medicine, this progress has taken its toll. It has been estimated that about one hundred and twenty-five lives have been lost during the progress of development of radiology. Many hundreds more have been terribly maimed. Through the suffering and death of these martyrs of science, the practice of radiology in the hands of competent operators is now as safe to the operator and to the patient as is any other branch of medicine. Our course, then, should include a short reference to these men and to their work.

The physics, mechanics, and technic of radiology are quite complicated, and we believe that any attempt to teach these to the undergraduate student is unwarranted. These details should be taught as postgraduate courses to those who are going into radiology as a specialty. A very short recital of the physical nature of the roentgen rays, their similarity to the gamma rays of radium, and the method of production of the rays is sufficient. This part of the course is given

in our institutions during a tour of inspection of the x-ray department. During this trip, the various mechanical appliances and their workings are explained without burdening the student with a tremendous amount of detail. The various protective devices, such as lead, barium plaster, lead glass, cork floor insulation and rubber gloves, are gone into in some detail, as a matter of future protection to the embryo physician and his still more embryonic patients, should he decide to put in a small office x-ray equipment. Roentgenologists see many patients and physicians who have been injured by these machines used by non-specialists, and hence it is our duty to warn the medical students of the potential sources of danger and the safeguards to be used.

By far the most important part of the course is that time devoted to the uses and limitations of radiology in diagnosis and therapeusis. We believe that the student should have had considerable didactic instruction in medicine, surgery, orthopedics, urology and other special subjects before taking the course in radiology. If this instruction has not already been received, the instructor may in many instances be demonstrating a condition of which the student has never even heard. There is also another very good reason for putting radiology rather late in the curriculum, and that is to avoid having the student in his own mind place an over-emphasis on this diagnostic agency. Our teaching hospital is perhaps no exception, and we have noticed an increasing tendency on the part of the interns to send every case down to the x-ray room as soon as the patient is admitted. In this way a short cut to diagnosis is attempted. A patient may be complaining of right upper quadrant pain, and a chest x-ray may show an old pleurisy in the right base; but the patient may have a definite peptic ulcer which may escape observation if the chest x-ray is made prior to the complete work up of the case, because a reason for the pain has already been found. A thorough training in history taking, physical diagnosis, and the actual handling of patients prior to the course in radiology will tend to combat this tendency and to show the proper interrelationship of the roentgen examination and other methods of diagnosis.

The group taking a course in radiology should not be too large, for it is our feeling that much more benefit may be derived from the small, rather intimate groups gathered around viewing boxes showing actual films, than from large classes, using lantern slides for demonstration. Seeing the actual detail on the film is of much importance, for this is the form in which the referring physician sees the pathological condition when he has called a roentgenologist in consultation. Lantern slides of x-ray films are better than nothing, but the detail obtained cannot compare with the original film. This detail cannot be seen if the group is so large that those in the rear are far away from the film shown.

The actual subject matter presented is well divided into various sections, such as bones and joints, gastrointestinal tract, urinary tract, and so on. In commencing a demonstration of any of these subdivisions, we first have a group of normals, for it is practically impossible to obtain a clear idea of a pathological process without first having a fairly good mental image of the normal. After the normals have been presented, the various diseases and abnormalities are shown, and an attempt is made properly to evaluate the roentgenologist's opinion concerning these conditions. The limitations of the roentgen ray as a diagnostic means are stressed just as forcibly as are its good features. A few concrete examples may be given. In hematogenous osteomyelitis, the fact is stressed that an early negative x-ray diagnosis is to be disregarded absolutely, since the demonstrable x-ray findings are seen only after a period of eight to fourteen days following the onset of the disease. In gall-bladder examinations, the point is made that, although the roentgenologist is correct in over 95 per cent of the cases reported as showing a non-functioning gall-bladder, he is accurate in only slightly over 80 per cent of the cases reported as normal. About 15 per cent of all calculi of the urinary tract cannot be demonstrated roentgenologically. These are only a few of the limitations which are presented. On the other hand, the advantages of this type of examination are also stressed. In the early exudative type of tuberculosis, where there is a small area of exudation just under the clavicle, the roentgen examination may give a definite, positive diagnosis in the complete absence of physical signs. In peptic ulcer and malignancy of the alimentary canal, the x-ray examination in the hands of well qualified men is without a peer as a diagnostic means. In this way, using the films as a text, the uses and limitations of roentgenology as a diagnostic agency are stressed. Relatively little is given concerning differential x-ray diagnosis, such as the fine points of difference between silicosis and tuberculosis, for we feel that these technical problems belong to the postgraduate curriculum. In the undergraduate years, we are not trying

to train specialists, but we are endeavoring to give a well-rounded, comprehensive idea of the science of medicine as a whole, giving enough detail so that the students may know whether the various branches appeal to them as possible fields for their life work. For the same reasons, we do not attempt to give all of the unusual conditions, for these may only cloud the major issues.

When the discussion of diagnosis has been finished, a relatively short period is devoted to radiation therapy, and a very candid criticism of its advantages and shortcomings is given, the emphasis being placed on the diseases which are amenable to radiation therapy, and the results which may be expected from this treatment. The matter of x-ray and radium burns is discussed, and the methods of protection and measurements of dosage are briefly given. The more recent advances in the treatment of infection, such as carbuncles and boils, are stressed, for it is in this field that radiation therapy is now extending its usefulness most rapidly.

After the completion of the purely didactic meetings, several periods are given to a little practical examination. The films which have been made of patients during the preceding day are brought in, and the students are taken in rotation and are asked to demonstrate the films, knowing only what the roentgenologist knew when the case was referred to his department. First, the student is asked to point out any deviations from normal, and then to try to diagnose these abnormalities after they have been ascertained. We have found that this is perhaps the most valuable part of the course. The students naturally profit by one another's mistakes, and these cases are remembered much more readily than the ones in which the diagnoses are made for them. Another good feature is that the rather high percentage of negative examinations is brought home more forcibly. During the didactic work, only positive cases have been shown and the student may have received the erroneous idea that an x-ray examination must always show an abnormality. Using the routine films, this impression is soon corrected.

To this regular course has recently been added an additional feature, which is apparently working quite well. The students in both the Out-Patient Dispensary and on the wards are now held responsible for going over the films of their patients with a medical member of the X-Ray Department. This seems to be helping considerably in correlating physical signs, history, and laboratory work, with what has been reported from the roentgen examination. Many times we are able to show why there is an apparent discrepancy between the x-ray and physical examinations. In pulmonary malignancy, the cause of the absence of physical signs may well be explained in the roentgen department, as it may well be shown by the roentgen examination that these large tissue masses, giving no dullness to percussion, are many times located in the central portions of the lungs, with normal lung tissue overlying them. On the other hand, a student may come down and go over a bleeding peptic ulcer case with us, after we have given a negative x-ray report. The reason for the failure of visualization of these small mucous erosions may then be easily explained, and the apparent discrepancy corrected. This part of the instruction is relatively new, but so far has proven quite satisfactory.

The foregoing is a rather brief outline of what we consider a fairly satisfactory course in roentgenology. The teaching has been changed and will be changed from time to time to keep pace with the advances of roentgenology and of the other medical subjects, but at present the general scheme as given seems to be sufficient.

SUMMARY

(1) Methods of teaching preclinical subjects by radiology are discussed.

(2) A brief outline of a course in radiology is given.

(3) This course should give only the uses and limitations of radiology, eliminating technical details.

(4) Special training in radiology should be a postgraduate study.

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TREATMENT OF NEUROSYPHILIS

Why about 10 per cent of syphilitic infections progress to a severe involvement of the central nervous system, while in the other 90 per cent of cases the organisms limit their destructive activities chiefly to other tissues, one does not know. Two possible hypotheses are tenable: a difference of bacterial strain, that is, a neurotropic or nerve loving organism may be the acquired infecting agent. Or there may be a diminished resistance of the nervous system of the host. The theory of the neurotropic spirochete has so far been unprofitable, prophylactically and therapeutically, since the syphilitic does not select his organism, and vaccines have no apparent effect on the course of the disease. Variations of resistance of the host probably account for the major variations in the clinical picture.

It is frequently stated that since the employment of arsphenamine the secondary protective skin reactions have been suppressed and neurosyphilis has consequently increased in incidence. Before the era of arsphenamine, according to Hyde,[1] the average incubation period of neurosyphilis was fifteen years; since arsphenamine, it has been shortened to seven. Some have claimed that neurosyphilitic manifestations occur only among inadequately treated cases, but this is by no means proven to be true. Modern understanding of lues has certainly not decreased the incidence of nerve involvement, and treatment of a diseased nervous system has been notoriously ineffectual. The most heroic measures of attack upon neurosyphilis of the past decade were the intraspinal injections of drugs or arsphenaminized serum which were employed for a time in the hope of deriving benefit from a direct contact of the antiseptics with treponema in the spinal cord and brain. These attempted direct sterilizations were, however, without results and have now been largely abandoned.[2]

In 1917, a curious innovation in the therapy of general paresis was instituted by von Jauregg. Because the inhabitants of tropical countries where sanitation is poor and the malaria rate high, rarely develop neurosyphilis, he gave inoculations of benign tertian malaria to patients in advanced stages of general paralysis. He reported considerable improvement of nearly one-third of these otherwise hopeless cases. There has since been some confirmation of his work, both clinical and pathological.[3]

Freeman[3] demonstrated that the number of spirochetes in the brain of general paretics actually diminishes following malarial inoculations. The indications for therapeutic malaria he now believes are broadening, and should include early tabes and other lesions of the spinal cord. Dennie,[4] of Kansas City, reports that destructive bone lesions of recent syphilitic origin will resolve under malarial therapy; and that interstitial keratitis and resistant skin lesions are likewise relieved. The plasmodium of malaria, he believes to be a most powerful stimulator of the reticulo-endothelial system.

The spirochetes of recurrent fever and ratbite fever have likewise been used in the treatment of syphilis. Diathermy[5] has been offered as a substitute for malaria by those who believe that malaria exerts its spirocheticidal effect by

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means of the high temperature which it induces. There is probably less general evidence of its effectiveness, since it has been less widely employed.

Hinsie and Blalock,[6] of the New York Psychiatric Institute, report a comparison of the results of treatment of general paresis with malaria alone, with tryparsamid alone, and with malaria and tryparsamid. They obtained their highest remission rate with tryparsamid alone, and this group of patients survived longest. Among the group which malaria had failed to benefit, tryparsamid produced a remission in 15 per cent of cases. The laboratory tests in all groups tended to become negative with time. The greatest incidence of negative findings occurred among those who received tryparsamid alone. There was, however, no parallelism between the clinical condition and the laboratory findings. It may be seen from this that the possibilities of accomplishment with drugs have not been exhausted, although these authors found certain cases distinctly benefited by the plasmodium.

Nicolle and Fitzgerald[1] likewise have studied the laboratory changes following malarial inoculations. They pointed out in 1926 that little change could be detected in the serological reactions within one year after malaria treatment, even in clinically improved cases. They have now re-examined these cases six to seven years after treatment with somewhat different findings. They observed in the spinal fluid a tendency toward normal reactions in the cell count, globulin, colloidal gold and other syphilitic tests. The serological improvement again, however, bore no relation to the clinical course.

It emerges in 1932 that neurosyphilis is occurring earlier and perhaps more frequently in the course of the disease than it has in the past; and that inoculations of a specific type of malaria seem to have a restorative effect, clinically and serologically, in a small number of cases. It would appear that some temerity might be required to inoculate an already sick man with a parasite which he need never have encountered. However, considering the severity and hopelessness of neurosyphilis, and the existing evidence of clinical improvement of a large total number of cases following malaria courses in various hands, it is probable that this means should be considered in any case of syphilis with beginning neurological manifestations. The patient should, however, give himself up entirely to his disease, as do the early tuberculous, and be institutionalized for continuous observation.

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THREE EXPERIMENTAL BLOOD DISEASES

The importance of small quantities of copper, first pointed out by Wisconsin workers,[1] upon milk anemia (caused by a diet of milk alone) is rather generally accepted by biochemists and clinicians. Meyer[2] and his coworkers at Western Reserve University still maintain that the effect is not specific for copper; that milk anemia may be repaired by iron alone or by iron plus one of several heavy metals, for example, arsenic, manganese, or zinc, or iron electrolytically free from copper,—even by the highly purified iron preparations of the Wisconsin investigators. However, the weight of expert testimony lends to copper an outstanding importance in blood building.

For clinical purposes, iron salts usually contain enough copper to prevent symptoms of this deficiency in infants who receive medicinal iron. However, occasionally babies who had become anemic upon prolonged milk feeding have apparently failed to improve when treated with iron alone, but showed an upward curve of hemoglobin production when small quantities of copper were added to the diet.

It is probable that feeding of copper in the usual therapeutic quantities is not harmful. There is no report that the red cells or hemoglobin are ever abnormally stimulated by it; they are merely under certain conditions restored to their normal quantity. There have been no reported cases of overproduction of red cells following copper or iron administration. Polycythemia is a disease which in human beings has some distressing manifestations. Its subjects are short lived. Its cause is not understood. The conventional treatment, bleeding and oxygen inhalations, is palliative and gives no lasting bene-

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fit. The experimental production of polycythemia would offer a new means of investigation of the disease. Orten[3] and others studied the effect upon the cell content of normal rat blood of a number of minerals and observed an abnormal, pronounced increase of the red cells, hemoglobin and cell volume when salts of cobalt were administered. These increases were not noted when other minerals, for example, manganese, nickel, zinc, copper and iron, were substituted. The leucocytes were not affected in any case. Only the red cells were stimulated, and only by cobalt. An experimental polycythemia of the red cells was thus induced.

At Peiping Union Medical College[4] it is reported that "thoratrast," a fluid containing thorium dioxide, is used for clinical pyelography and is also given intravenously to outline the liver and spleen. Usually the only noticeable ill effects which have followed its use were a transient anemia and a leucocytosis. Shih and Jung, upon giving it to rabbits intravenously, report an extreme reduction of the platelets, a delayed bleeding time, weakness and extravasation of blood into various organs. Following larger doses, there were extensive hemorrhages. "Thoratrast," chiefly through its thorium content they say, lowers the thrombocytes of the blood and produces acute purpura hemorrhagica.

Thus three of the well known blood diseases of mankind: (1) secondary anemia or shortage of red blood cells through failure of production; (2) polycythemia, or a pathological oversupply of red cells through overstimulation of the red cell producing mechanism; and (3) a diminution of the platelets, accompanied by purpura hemorrhagica, or extravasation of blood outside the veins and arteries, may be induced by the presence or absence of small quantities of salts of three heavy metals, copper, cobalt, and thorium. Copper is therapeutically useful. A place among the beneficial drugs is possibly to be found for cobalt. Thorium, one would infer for the present, is to be as far as possible avoided.

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GLEANINGS FROM RECENT JOURNALS

Malignancy and Radioactivity.—The general public has followed the history of the doomed young women who twelve years or more ago were employed in a New Jersey factory to paint illuminated watch dials. A radioactive material was used which they ingested by pointing the brushes with their lips, and which accumulated in their bodies. Several types of fatal symptoms have since slowly developed in them. Necrosis of the jaw was the first abnormality noted, usually by dentists, in those who had been employed even for a brief period. There was later a profound weakness and anemia, and a characteristic rarefaction of the bones was revealed by the x-ray.[1] A number of deaths have occurred. By 1928, Martland, who studied many of the patients, believed that the worst period was over, although most of the former dial painters were still radioactive. However, eight or more cases of a rapidly growing osteogenic sarcoma in those who have survived this long period have recently been brought to light.[2] There is of course no means of foretelling the ultimate effects upon the remaining poisoned individuals of the disintegration of radioactive elements *in vivo*. Slow bone destruction, blood destruction, and this rare, late developing sarcoma are particularly noted.

A new means of experimental production of malignancy by radioactive elements has thus accidentally been demonstrated. According to Martland, such sarcomata in animals could probably not be developed rapidly by heavy radiation. Slow bombardment of the tissues by minute quantities of these elements over long periods of time is essential. Malignant degeneration of x-ray burns also occurs more frequently following many exposures over a long period of time than following a single massive dose or a few doses at short intervals.[3]

It is suggested that other forms of malignancy in the body may be dependent on the presence of concentrations of radioactive substances too small to be detected by present methods.[2] There seems to be, however, scant evidence for this belief. The radium sarcomas would appear to do no more than confirm the

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2. Malignant Conditions in Radioactive Persons. Editorial, J.A.M.A., 97:1968, Dec. 26, 1931.

3. Davis, J. S.: Deep Roentgen Ray Burns, with a Note on Burns Caused by Radium. Amer. Jour. Roentgenol. & Ra. Ther., 26:890, Dec., 1931.

hypothesis that prolonged irritation, not too severe, and of many types, is the cause of malignancy. One may go so far as to say that the watch dial sarcomas deal another blow to the already moribund theory of specific bacterial causation of malignant growths; they add a prop to the hypothesis of chronic irritation.

Obviously, patent radioactive drugs which are reported to be employed for various ailments[2] are to be condemned, and should be forbidden by legislation.

Multiple Sclerosis.—Weil[4] reports on the efforts of English bacteriologists to prove that a filterable virus is the cause of this disease. Culture media and methods of ultraviolet photography of bacterial colonies similar to those which Barnard and Gye employed in attempting to show that a filterable virus causes cancer have been used. Weil, repeating the work, says that the same photographic appearance of bacterial colonies with spheres and granules and filaments, like the reputed ones of multiple sclerosis, may be obtained from spinal fluids of tuberculosis, hyperthyroidism or from boiled spinal fluid. He believes that the globulin content of the spinal fluid is responsible for the picture, and that no filterable virus of multiple sclerosis has been demonstrated. The cause of multiple sclerosis, he says, has not been proven to be bacterial.

To show that there is a specific myelolytic toxin in the urine of patients suffering from the disease, he has incubated concentrated urines of multiple sclerosis with rat spinal cords. The myelin sheath of the cord was attacked by nearly all the urines of multiple sclerosis and by an occasional normal urine. The destruction, Weil believed, closely resembled that of the spinal cords of human multiple sclerosis.

Putnam, McKenna and Morrison[5] say that the same pathological condition of the myelin sheath of living animals follows various types of injury which result in obstruction of a relatively long stretch of a relatively large blood vessel. It is not necessary to postulate a specific virus, toxin, or ferment to account for the histological appearance of multiple sclerosis.

English Medical Comment on Football.[6]—"American football demands from its votaries so many hours of attentive study with a professor of football tactics (called a coach)," says the British *Lancet,* following a discussion of the gridiron fatalities of the year, "and such exercise of memory that one marvels that it should be classed as a recreation. Perhaps the co-educational system is a factor in its popularity. For there is no doubt that a good football player exhibits qualities both of strength and of skill that endear him to the other sex. Perhaps, too, these very qualities rank higher in the scale of virtues adopted by a young nation than they do among the more sophisticated nations of Europe."

Now, when times are hard, and Americans feel possibly a little care-worn and middle-aged, this foreign reminder of the innocence and ingenuousness of their pleasures may be gratifying.

International Conference on Birth Control.—Little authentic information upon contraception has been available in the United States. The physician who has advised a nephritic or tuberculous patient not to bear children has found few scientific sources from which to draw information as to the best means of prevention. His knowledge has frequently been gained either from agents selling contraceptive devices, or by word of mouth from other physicians. A critical survey of the practice of contraception in America and foreign countries has recently been published.[7]

The various methods in use over a number of years are reviewed by their proponents and discussed by attending authorities. An occlusive pessary used in the early Eighteenth Century, according to Dr. Stone, was the scooped out half of a lemon, which was placed over the cervix. Modern methods described and evaluated are: the mechanical occlusive, with description of many appliances in use; the intrauterine; the chemical; the biological, which includes a brief report on studies of hormonic control of conception, and of immunization against spermatozoa; methods of sterilization; and methods of inducing abortion. There are carefully compiled series of cases with statistics of birth control clinics from the United States, England, Germany, Russia, Austria, Holland and Denmark, India, China, and Japan.

4. Weil, Arthur: A Study of the Etiology of Multiple Sclerosis. J.A.M.A., 97:1587, Nov. 28, 1931.
5. Putnam, T. J.; McKenna, J. B.; and Morrison, L. R.: Studies in Sclerosis: Histogenesis of Experimental Sclerotic Plaques and Their Relation to Multiple Sclerosis. Idem., p. 1591.
6. U. S. Correspondent, Lancet, 222:100, Jan. 9, 1932.
7. The Practice of Contraception. An international symposium and survey. Edited by Margaret Sanger and Hannah M. Stone, M.D. With a foreword by Robert L. Dickinson, M.D. From the proceedings of the Seventh International Birth Control Conference, Zurich, Switzerland, September, 1930. Baltimore: The Williams & Wilkins Company, 1931.

Typical of the suspicion and misunderstanding current between Japan and the United States, and in fact of the hostile interpretation of every new idea by the masses of every race, is the report of the physician from Tokyo, of the attitude of some of his compatriots toward Margaret Sanger when she first visited Japan with her propaganda for birth control. It was said that she was working to diminish the population of Japan in order to make it easier for the United States to conquer the country.

Contraception is a phase of modern medicine which physicians treating women patients cannot afford to neglect.

Book Reviews

Yellow Fever: An Epidemiological and Historical Study of Its Place of Origin. By Henry Rose Carter, Late Assistant Surgeon General, United States Public Health Service. Edited for Publication by Laura Armistead Carter and Wade Hampton Frost. 308 pages. Baltimore: The Williams & Wilkins Company, 1931. Cloth, $5.00.

Carter's "History of Yellow-Fever" more nearly than any work shows his full mastery of the subject.

It is characteristic in its plan, in that he first lays down, from modern knowledge, the physical, biological and social conditions which determine the characteristic epidemiology of yellow-fever. Then, in the light of these conditions, he reviews not the history of yellow-fever, but the history of the period from the earliest records of Western Africa and America to 1648, when yellow-fever emerges from obscurity and becomes recognizable beyond question. In all that may throw light on yellow-fever, as in the old Maya and Aztec records, in the details of losses in military expeditions and colonies, in the analysis of social conditions and commercial development, in the study of early communication between Africa and America, he has gone back to contemporary records . . . and his interpretation of the facts with reference to the light which they throw on the presence or absence of yellow-fever is the most delightful epidemiological analysis the reader has ever followed. Merely as an historical research his history of yellow-fever would be a credit to an historian. As a retrospective study in epidemiology it is altogether unique and it is believed that it will be final as to the principal questions which he discusses.

Proceedings of the Twenty-Fifth Annual Conference of the American Association of Medical Milk Commissions, Inc., and the Certified Milk Producers' Association of America, Inc. 334 pages with tables, charts and reports. New York: The American Association of Medical Milk Commissions, Inc., 1931.

Among the subjects of medical interest discussed are the pasteurization of certified milk, calcium metabolism, septic sore throat, and control of mastitis.

A Text-Book of Clinical Neurology. By Israel S. Wechsler, M.D., Professor of Clinical Neurology, Columbia University, New York; Attending Neurologist, Neurological Institute and the Montefiore Hospital, New York. Second Edition, Revised. 759 pages with illustrations. Philadelphia: W. B. Saunders Company, 1931. Cloth, $7.00.

The opening chapters deal with methods of examination and history taking. The classification of diseases is anatomical and there are sections dealing with the abnormalities of the spinal cord, of the peripheral nerves, the brain, and with the neuroses. A chapter upon psychometric tests is included, which correctly evaluates this aid to neurological examination.

In general, the diseases which are classified as neurological are treated upon a physiological rather than upon a psychiatric basis, which is a distinct advantage. It is an excellent reference or text-book.

Modern Medical Treatment. Volumes I and II. By E. Bellingham-Smith, M.D., F.R.C.P. (Lond.), Physician and Lecturer on Medicine, St. George's Hospital, and Anthony Feiling, M.D. (Camb.), F.R.C.P. (Lond.). With an Introduction by Sir Humphry Rolleston, Bart., G.C.V.O., K.C.B. 1,406 pages with tables and charts. New York: William Wood & Co., 1931. Cloth, $12.00 per set.

The arrangement of this work is rather confusing, as diseases are not grouped strictly accordingly to etiology. For example, migraine and other headaches have one section. There is another on insomnia.

The sections on anemia are entirely inadequate. There is no mention of the recently isolated active suprarenal extract in the chapter on Addison's disease; instead, various prescriptions of bismuth or charcoal for combating the accompanying diarrhea are the chief therapeutic suggestions. Sanocrysin is advocated for the treatment of tuberculosis.

The title is somewhat misleading, as the treatments described are not particularly modern. One's library fund could be much better invested than in these rather expensive tomes.

Southern Medical News

ALABAMA

Dr. M. Toulmin Gaines, Mobile, has been appointed the member of the Council from Alabama of the Southern Medical Association, the appointment having been announced recently by the President, Dr. Lewis J. Moorman, Oklahoma City, Oklahoma. Dr. Gaines succeeds Dr. W. W. Harper, Selma, whose term had expired, and who having served the constitutional limit, was not eligible for reappointment.

The Southeastern Surgical Congress will hold its third annual assembly in Birmingham, March 7 and 8, the Tutwiler Hotel being assembly headquarters. Dr. C. W. Roberts, Atlanta, Georgia, is President, Dr. Frank K. Boland, Atlanta, Georgia, is President-Elect, Dr. B. T. Beasley, Atlanta, Georgia, is Secretary, and Dr. Gilbert F. Douglas, Birmingham, is General Chairman for the Birmingham assembly.

The Bulloch County Medical Society has elected the following officers for 1932: Dr. G. M. Guthrie, Inverness, President; Dr. J. W. Thomason, Midway, Vice-President; and Dr. J. K. Haygood, Union Springs, Secretary-Treasurer.

MEDICAL NEWS continued on page 323

OFFICERS, SOUTHERN MEDICAL ASSOCIATION, 1931-1932

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Section on Urology—Dr. Russell A. Hennessey, Chairman, Memphis, Tennessee; Dr. Neil S. Moore, Vice-Chairman, St. Louis, Missouri; Dr. Earl H. Floyd, Secretary, Atlanta, Georgia.

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Section on Public Health—Dr. J. D. Applewhite, Chairman, Macon, Georgia; Dr. Joseph W. Mountin, Vice-Chairman, Washington, D. C.; Dr. Leon Banov, Secretary, Charleston, South Carolina.

National Malaria Committee (meeting conjointly with Southern Medical Association)—Dr. L. O. Howard, Honorary Chairman, Washington, D. C.; Dr. E. L. Bishop, Chairman, Nashville, Tennessee; Dr. Charles F. Craig, Chairman-Elect, New Orleans, Louisiana; Dr. Henry Hanson, Vice-Chairman, Jacksonville, Florida; Dr. Mark F. Boyd, Secretary, Tallahassee, Florida.

American Society of Tropical Medicine (meeting conjointly with Southern Medical Association, 1932)—Dr. Frank Smithies, President, Chicago, Illinois; Dr. E. L. Walker, First Vice-President, San Francisco, California; Dr. G. R. Callender, Second Vice-President, Washington, D. C.; Dr. Henry E. Meleney, Secretary-Treasurer, Nashville, Tennessee; Dr. Damaso de Rivas, Assistant Secretary, Philadelphia, Pennsylvania.

Section on Medical Education—Dr. Kenneth M. Lynch, Chairman, Charleston, South Carolina; Dr. John Walker Moore, Vice-Chairman, Louisville, Kentucky; Dr. Ernest W. Goodpasture, Secretary, Nashville, Tennessee.

Southern Association of Anesthetists (meeting conjointly with Southern Medical Association)—Dr. H. Boyd Stewart, President, Tulsa, Oklahoma; Dr. Mary E. Hopkins, First Vice-President, Louisville, Kentucky; Dr. Dennis P. Harris, Second Vice-President, Beaumont, Texas; Dr. W. Hamilton Long, Secretary-Treasurer, Louisville, Kentucky.

Women Physicians of Southern Medical Association—Dr. Kate Savage Zerfoss, Chairman, Nashville, Tennessee; Dr. Maud Loeber, Vice-Chairman, New Orleans, Louisiana; Dr. Lillian H. South, Secretary, Louisville, Kentucky.

Woman's Auxiliary to the Southern Medical Association—Mrs. Charles E. Oates, President, North Little Rock, Arkansas; Mrs. A. A. Herold, President-Elect, Shreveport, Louisiana; Mrs. S. E. Driskell, First Vice-President, Jacksonville, Florida; Mrs. S. M. Blackshear, Second Vice-President, New Orleans, Louisiana; Mrs. J. Bonar White, Recording Secretary, Atlanta, Georgia; Mrs. M. J. Kilbury, Corresponding Secretary, Little Rock, Arkansas; Mrs. Mabel Mason, Treasurer, Lumberton, Mississippi; Mrs. S. A. Collom, Jr., Historian, Texarkana, Texas; Mrs. J. D. Perdue, Parliamentarian, Mobile, Alabama.

JEFFERSON COUNTY MEDICAL SOCIETY

Birmingham, Alabama

HOST to the SOUTHERN MEDICAL ASSOCIATION, Twenty-Sixth Annual Meeting, November 16-18, 1932

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Transportation—Dr. J. A. Meadows
Golf—Dr. A. B. Harris
Trap Shooting—Dr. A. L. Atwood
Women Physicians—Dr. Melson Barfield-Carter
Ladies' Entertainment—Woman's Auxiliary, Mrs. John M. Akin, President; Mrs. Sid W. Collier, Chairman

Southern Medical News

Continued from page 321

Calhoun County Medical Society has elected the following officers for the ensuing year: Dr. John D. Durden, Anniston, President; Dr. B. F. Caffey, Choccolocco, Vice-President; Dr. C. Hal Cleveland, Anniston, Secretary; and Dr. E. C. Anderson, Anniston, Treasurer.

Colbert County Medical Society has elected the following officers for the coming year: Dr. W. A. Finley, Cherokee, President; Dr. H. A. Griffith, Sheffield, Vice-President; and Dr. J. P. Long, Sheffield, Secretary-Treasurer.

Cullman County Medical Society has elected the following officers for the coming year: Dr. R. A. Culpepper, Cullman, President; Dr. J. W. Wood, Hanceville, Vice-President; and Dr. M. S. Whiteside, Cullman, Secretary-Treasurer.

Etowah County Medical Society has elected the following officers for 1932: Dr. C. L. Murphree, Gadsden, President; Dr. J. M. McElroy, Attalla, Vice-President; and Dr. DeWitt Faucett, Gadsden, Secretary-Treasurer.

Geneva County Medical Society has elected the following officers for 1932: Dr. Ernest Tankersley, Samson, President; Dr. C. P. Gay, Geneva, Vice-President; and Dr. M. E. Doughty, Slocomb, Secretary-Treasurer.

Houston County Medical Society has elected the following officers for 1932: Dr. J. T. Ellis, Dothan, President; Dr. C. W. Hilliard, Dothan, Vice-President; and Dr. F. G. Granger, Dothan, Secretary-Treasurer.

Jackson County Medical Society has elected the following officers for 1932: Dr. Hugh Boyd, Scottsboro, President; Dr. J. A. Gentry, Stevenson, Vice-President; and Dr. M. H. Lynch, Scottsboro, Secretary-Treasurer.

Lamar County Medical Society has elected the following officers for 1932: Dr. G. S. Barksdale, Fernbank, President; Dr. R. H. Redden, Sulligent, Vice-President; and Dr. J. A. Jackson, Vernon, Secretary-Treasurer.

Lauderdale County Medical Society has elected the following officers for 1932: Dr. A. A. Jackson, Florence, President; Dr. W. R. Moore, Florence, Vice-President; and Dr. W. D. Hubbard, Florence, Secretary-Treasurer.

Mobile County Medical Society has elected the following officers for 1932: Dr. J. H. Dodson, Mobile, President; Dr. J. Mac Bell, Mobile, Vice-President; Dr. W. W. Scales, Mobile, Secretary; and Dr. J. H. Little, Mobile, Treasurer.

Tuscaloosa County Medical Society has elected the following officers for 1932: Dr. J. Henry Somerville, President; Dr. R. C. Partlow, Vice-President; and Dr. Ralph McBurney, Secretary-Treasurer; all of Tuscaloosa.

Dr. W. D. Hubbard, Florence, has been elected County Health Officer for Lauderdale County.

Dr. M. Toulmin Gaines, Mobile, was elected Librarian of Mobile County Medical Society.

Dr. G. A. Cryer, Anniston, has been elected County Health Officer for three years.

Dr. Robert Briggs Beard, Troy, and Miss Lois Adams were married January 2.

Deaths

Dr. Gottlob Hartung, Cullman, aged 88, died December 18.

Dr. Thomas Young Greet, Gadsden, aged 50, died December 12.

Dr. M. R. Seay, Fernbank, aged 79, died December 10 of edema of the lungs.

Dr. St. John Naftel, Lapine, aged 75, died December 15 of pneumonia.

Dr. William John Hannah, Montgomery, aged 72, died January 2.

Dr. William Ira Wright, Collinsville, aged 68, died December 2 of angina pectoris.

Dr. William Wesley Clapp, Birmingham, aged 85, died December 13 of lobar pneumonia.

Dr. James Heustis Jones, Camden, aged 54, died February 2.

Dr. J. M. Watkins, Troy, aged 62, died in February.

ARKANSAS

Dr. Morgan Smith, Little Rock, has been reappointed the member of the Council from Arkansas of the Southern Medical Association, the reappointment having been announced recently by the President, Dr. Lewis J. Moorman, Oklahoma City, Oklahoma.

Pulaski County Medical Society has elected the following officers for 1932: Dr. H. Fay H. Jones, Little Rock, President; Dr. S. C. Fulmer, Little Rock, Vice-President; Dr. Ernest H. White, Little Rock, Treasurer; and Dr. William R. Bathurst, Little Rock, Secretary.

Sebastian County Medical Society has elected the following officers for 1932: Dr. E. C. Moulton, Fort Smith, President; Dr. B. L. Ware, Greenwood, Vice-President; Dr. J. W. Amis, Fort Smith, Secretary; and Dr. W. R. Brooksher, Jr., Fort Smith, Treasurer.

Dr. R. M. Eubanks, Little Rock, is the newly elected President of the State Board of Health.

Dr. Howard Rand has succeeded Dr. Alphonso Isom, Dumas, in charge of the Dumas Hospital.

Dr. W. H. Horn and Dr. T. S. Jordan, both of Taylor, have recently opened a sanitarium in Magnolia.

At a meeting in January of the Jefferson County Medical Society a Fourth Councilor District Medical Society was organized, with the following elected as officers: Dr. J. S. Wilson, Monticello, President; Dr. H. T. Smith, McGehee, Vice-President; and Dr. W. T. Lowe, Pine Bluff, Secretary-Treasurer. Monticello was selected as the first meeting place.

Deaths

Dr. Charles Francis Thomas, Fordyce, aged 62, died December 19.

Dr. James H. Higgins, Altus, aged 60, died October 16.

Dr. Arch Sylvester Chapman, Fort Smith, aged 46, died December 22.

Dr. William Edward Gray, Little Rock, aged 54, died January 10.

Dr. Zilliba L. Kirkham, Little Rock, aged 64, died December 29.

Dr. William Enoch Jones, Little Rock, aged 62, died December 25.

Dr. Charles B. Johnston, Harrell, aged 60, died November 17.

DISTRICT OF COLUMBIA

Dr. Hugh S. Cumming, Surgeon General, U. S. Public Health Service, Washington, has been reappointed and confirmed to the office of Surgeon General another term of four years.

Dr. William Gerry Morgan, Washington, has been reappointed a member of the Council from the District of Columbia of the Southern Medical Association, the reappointment having been announced recently by the President, Dr. Lewis J. Moorman, Oklahoma City, Oklahoma.

Dr. Hugh S. Cumming, Surgeon General, U. S. Public Health Service, Washington, was a guest speaker of the Richmond Chapter of the University of Virginia Alumni Association at its meeting in January.

The George Washington Medical Society, Washington, had as their guest speaker at a recent meeting Dr. Robert L. Payne, Norfolk.

Deaths

Dr. Verner Trenary Scott, Major, M.C., U. S. Army, Washington, aged 43, died December 15.

Dr. Nelson Gapen, Colonel, U. S. Army, retired, Washington, aged 53, died January 7.

FLORIDA

Dade County Medical Society has elected the following officers for 1932: Dr. C. F. Roche, Miami Beach, President; Dr. Elmo D. French, Miami, Vice-President; Dr. Robert T. Spicer, Miami, Secretary; and Dr. Frank Davis, Miami, Treasurer.

Hillsboro County Medical Society has elected the following officers for 1932: Dr. L. F. Carlton, Tampa, President; Dr. E. S. Gilmer, Tampa, Vice-President; and Dr. J. T. Cowart, Tampa, Secretary-Treasurer.

Palm Beach County Medical Society has elected the following officers for 1932: Dr. L. J. Netto, West Palm Beach, President; Dr. J. L. Carlisle, West Palm Beach, Vice-President; Dr. V. M. Johnson, West Palm Beach, Secretary; and Dr. F. K. Herpel, West Palm Beach, Treasurer.

Pasco-Hernando-Citrus County Medical Society has elected the following officers for 1932: Dr. A. B. Cannon,, Lacoochee, President; Dr. Leland H. Dame, Inverness, and Dr. L. T. Furlow, Brooksville, Vice-Presidents; and Dr. G. R. Creekmore, Brooksville, Secretary-Treasurer.

Dr. Walter D. Webb, St. Augustine, has moved to Boca Grande to become house physician for the Boca Grande Inn, Gasparilla Inn, and Useppa Inn.

Dr. Robert E. Baldwin, Tampa, has been appointed Superintendent of the Tampa Municipal Hospital, succeeding W. I. Webb, acting Superintendent.

Dr. C. J. Heinberg, Pensacola, is the newly elected first Vice-President of the Pensacola Kiwanis Club.

Dr. L. S. Laffitte, Gulf Hammock, is doing special work in urology in Philadelphia.

Dr. A. L. Rowe, Lake Worth, has completed two years of postgraduate work in urology and has resumed his practice in Lake Worth.

Dr. Wilbur O. Arnold, West Palm Beach, has completed a four-month postgraduate course at the University of Vienna.

Dr. R. H. Williams, Eustis, has resumed his practice in Eustis after doing postgraduate work in Vienna.

Dr. Charles Frederick Henley, Jacksonville, and Miss Ruby Belle Chambers were married in November.

Deaths

Dr. Sylvan McElroy, Orlando City, aged 48, died suddenly January 1.

Dr. George Henry Bowen, St. Petersburg, aged 76, died January 8 of carcinoma of the liver and pancreas.

Dr. Robert Lee Bryans, Pensacola, aged 67, died suddenly November 18.

GEORGIA

Bartow County Medical Society has elected the following officers for 1932: Dr. W. E. Wofford, Cartersville, President; Dr. T. Lowry, Cartersville, Vice-President; and Dr. A. C. Shamblin, Cartersville, Secretary-Treasurer.

Fulton County Medical Society has elected the following officers for 1932: Dr. W. E. Barber, Atlanta, President-Elect; and Dr. Trimble Johnson, Atlanta, Vice-President.

The Georgia Medical Society (Chatham County), has elected the following officers for 1932: Dr. William A. Cole, President; Dr. Robert Drane, President-Elect; Dr. John L. Elliott, Vice-President; and Dr. Otto M. Schwalb, Secretary-Treasurer; all of Savannah.

Thomas County Medical Society has elected the following officers for 1932: Dr. Harry Ainsworth, Thomasville, President; Dr. J. I. Palmer, Thomasville, Vice-President; and Dr. H. M. Moore, Thomasville, Secretary-Treasurer.

Troup County Medical Society has elected the following officers for 1932: Dr. W. P. Phillips, President; and Dr. J. E. Lane, Secretary-Treasurer, both of LaGrange.

Washington County Medical Society has elected the following officers for 1932: Dr. B. L. Helton, Sandersville, President; Dr. L. C. Mitchell, Sandersville, Vice-President; and Dr. W. M. Cason, Sandersville, Secretary-Treasurer.

Wilcox County Medical Society has elected the following officers for 1932: Dr. S. R. Mitchell, President; and Dr. J. T. Gammage, Secretary-Treasurer; both of Pineville.

The Medical Association of Georgia will hold its annual meeting in Savannah May 17-20.

The Fulton County Pediatric Society has elected the following officers for 1932: Dr. T. Bolling Gay, Atlanta, President; Dr.

Continued on page 32

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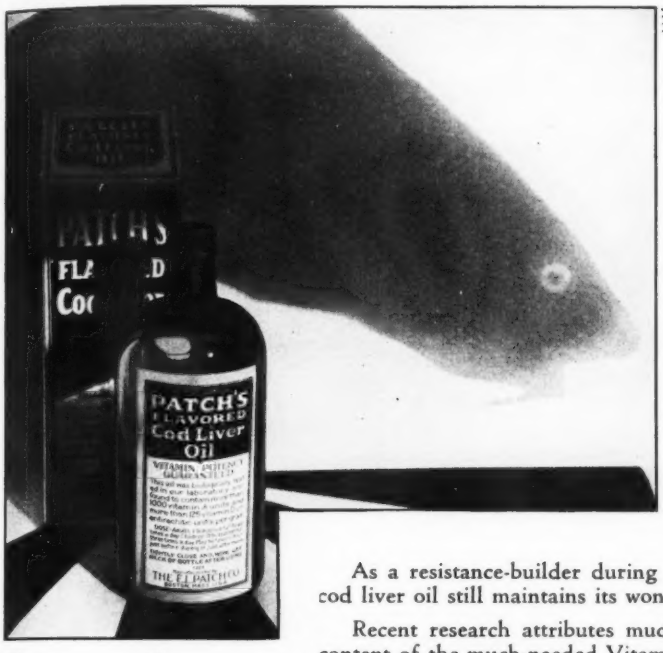

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Dr.....

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.....

Continued from page 324

Joseph Yampolsky, Atlanta, First Vice-President; Dr. H. Homer Allen, Decatur, Second Vice-President; and Dr. Roger W. Dickson, Atlanta, Secretary-Treasurer.

Dr. J. R. McGibony, formerly of Greensboro, has moved to Belcourt, North Dakota.

Dr. Virgil W. Osborne, Atlanta, has moved into new offices in the Dostors Building, where he will limit his practice to diseases of children and infant feeding.

Dr. J. Calvin Weaver, Atlanta, announces that he will limit his practice to surgery of the brain and spinal cord, and to organic diseases of the nervous system.

Dr. Logan Thomas, Dawson, is the newly elected official surgeon for the W. C. Davis Camp No. 15, United Spanish War Veterans.

DEATHS

Dr. Ivy Thompson Goldin, Cedartown, aged 47, died December 2 of influenza.

Dr. James Nottingham Carter, Savannah, aged 53, died December 6 of heart disease.

Dr. Samuel D. Warnock, Atlanta, aged 63, died November 30.

Dr. Kirby S. Williams, Thomaston, aged 67, died December 13.

Dr. Alonza Terrell Ray, Sharon, aged 61, died November 24.

Dr. Henry R. Butler, Atlanta, aged 71, died December 17.

Dr. Lawrence R. Brown, Sharon, aged 69, died November 26.

Dr. Charles F. McLain, Calhoun, aged 64, died December 26.

KENTUCKY

Bourbon County Medical Society has elected the following officers for 1932: Dr. J. C. Hart, Paris, President; Dr. William Kenney, Paris, and Dr. H. M. Boxley, Millersburg, Vice-Presidents; and Dr. M. J. Stern, Lexington, Secretary-Treasurer.

Franklin County Medical Society has elected the following officers for 1932: Dr. George H. Heilman, President; Dr. M. C. Darnell, Vice-President; and Dr. C. E. Youman, Secretary-Treasurer; all of Frankfort.

Grant County Medical Society has elected the following officers for 1932: Dr. J. W. Abernathy, Williamstown, President; Dr. A. D. Blaine, Dry Ridge, Vice-President; and Dr. C. A. Eckler, Dry Ridge, Secretary-Treasurer.

McCracken County Medical Society has elected the following officers for 1932: Dr. Bob Overbey, President; Dr. Palmer H. Reed, Vice-President; Dr. Leon Higdon, Secretary; and Dr. E. W. Jackson, Treasurer; all of Paducah.

The Licking Valley Medical Society has elected the following officers for 1932: Dr. W. B. Moore, Cynthiana, President; Dr. Hadley Caldwell, Newport, Dr. J. C. Norris, Bracken County, and Dr. N. P. Ellis, Williamstown, Vice-Presidents; and Dr. Edward Nelson, Falmouth, Secretary-Treasurer.

DEATHS

Dr. B. O. Rand, Milton, aged 60, died November 21.

Dr. Howe H. Wallace, Hopkinsville, aged 72, died December 11.

Continued on page 34

NEW! NEW!

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"As modern as tomorrow with prices of today"

A new set of treatment room furniture of startling beauty in design and finish. Many built-in conveniences to facilitate your work. See this beautiful new furniture at your nearest dealer today.

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A NEW BEAUTY—
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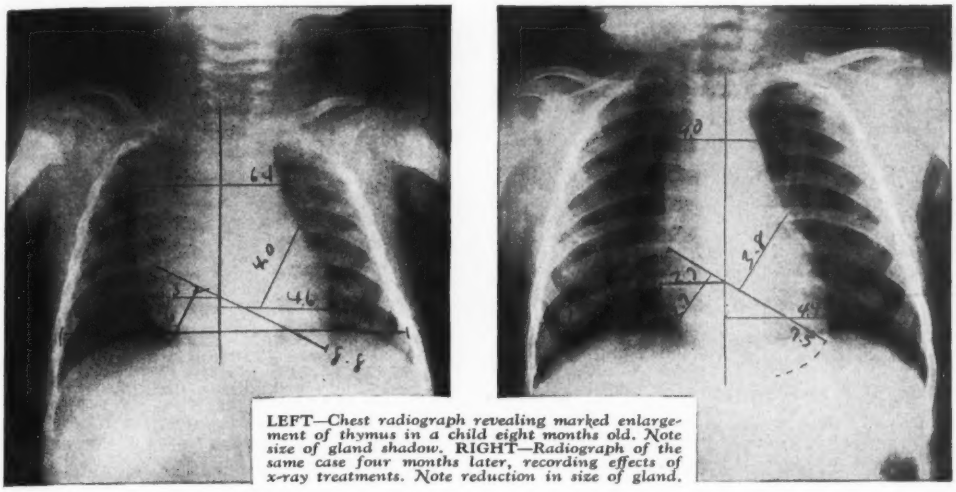
X-ray Examination Demonstrates *the Persistent Thymus*



LEFT—Chest radiograph revealing marked enlargement of thymus in a child eight months old. Note size of gland shadow. RIGHT—Radiograph of the same case four months later, recording effects of x-ray treatments. Note reduction in size of gland.

VERY young infants and children frequently exhibit signs of an over-active thymus. Statistics indicate that more than 7% of all infants have enlarged thymus; that between 5% and 7% of children of adenoid age show x-ray shadows suggestive of thymus hypertrophy. Such a condition may result in death, if the child is anesthetized.

Radiography offers a simple, economical, and certain method of detecting the persistent thymus in time to prevent sudden death; a few x-ray treatments are usually sufficient to eliminate the danger. Because of the serious complications that may arise from thymus hypertrophy, *routine radiography* should be a requirement in your practice.

* * *

In radiography of infants and children, film sensitivity is very important. The shortest possible exposure factor is necessary, due to difficulty usually encountered in immobilizing the patient and temporarily suspending respiration. Eastman Ultra-Speed and Eastman Diaphax X-ray Films present extreme sensitivity. In addition, they are uniform, facilitating comparisons between radiographs taken before and after treatment. Your radiologist can provide the highest quality radiographs with these films.

Radiography and clinical photography are indispensable in modern medical service. Send the coupon for the free bi-monthly magazine devoted to these subjects.

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Gentlemen: Please place my name on your free mailing list to receive "Radiography and Clinical Photography," regularly.

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City and State.....

Continued from page 32

Dr. William Edward Applehaus, Louisville, aged 32, died December 16.

Dr. Thomas J. Winder, Springdale, aged 79, died January 3.

Dr. James J. Booker, Greensburg, aged 81, died November 28.

Dr. Aquilla H. Durrum, Myers, aged 73, died December 7.

LOUISIANA

Caddo Parish Medical Society has elected the following officers for 1932: Dr. B. C. Garrett, President; Dr. William B. Heidorn, Secretary; and Dr. J. E. Knighton, Jr., Treasurer; all of Shreveport.

East Baton Rouge Parish Medical Society has elected the following officers for 1932: Dr. Rufus Jackson, President; Dr. T. C. Foreman, Vice-President; and Dr. W. H. Pipes, Secretary-Treasurer; all of Baton Rouge.

Morehouse Parish Medical Society has elected the following officers for 1932: Dr. E. L. Miller, Bonita, President; Dr. J. N. Jones, Bastrop, Vice-President; and Dr. L. E. Larche, Bastrop, Secretary-Treasurer.

Rapides Parish Medical Society has elected the following officers for 1932: Dr. L. D. Gremillion, President; Dr. S. J. Phillips, First Vice-President; Dr. E. R. Gandy, Second Vice-President; and Dr. D. B. Barber, Secretary-Treasurer; all of Alexandria.

Vernon Parish Medical Society has elected the following officers for 1932: Dr. W. E. Reid, President; Dr. W. W. Talbot, Vice-President; and Dr. D. O. Willis, Secretary-Treasurer; all of Leesville.

Washington Parish Medical Society has elected the following officers for 1932: Dr. H. V. Jones, Bogalusa, President; Dr. L. L. Lancaster, Bogalusa, Vice-President; and Dr. F. A. Williams, Franklinton, Secretary-Treasurer.

Dr. C. Jeff Miller, New Orleans, delivered the tenth annual Hodgen Lecture before the St. Louis Surgical Society in January.

BACILLUS ACIDOPHILUS CULTURE
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It is a significant fact that many physicians who prescribed B. B. CULTURE twenty-one years ago are still specifying B. B. CULTURE and now BACILLUS ACIDOPHILUS CULTURE (B. A. CULTURE) as well.

This ability to successfully meet the test of the years is a result of satisfactory service, a service which is no more distant than the nearest prescription pharmacy.

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French Hospital, New Orleans, has the following officers for 1932: Dr. H. B. Alsobrook, Chairman; Dr. S. C. Lyons, Vice-Chairman; and Dr. C. J. Brown, Secretary, all of New Orleans.

MARYLAND

Dr. Dudley Pleasants Bowe, Baltimore, has moved into new offices on Read Street.

Dr. W. T. Harrison, U. S. Public Health Service, Washington, recently delivered an address to the health officers of the State of Maryland at Baltimore.

DEATHS

Dr. James Edward Deets, Clarksburg, aged 75, died December 22.

Dr. Samuel Lunt Hannon, White Plains, aged 73, died December 24 in Walter Reed Hospital, Washington.

Dr. Oliver Newell Duvall, Baltimore, aged 63, died December 12 of acute myocarditis.

Dr. Nathan Alpert, Baltimore, aged 49, died December 14 of heart disease.

Dr. John Gilbert Selby, Cumberland, aged 59, died November 28 of pneumonia following an operation.

Dr. William T. Pratt, Rockville, aged 63, died December 16 of heart disease.

MSSISSIPPI

The Central Medical Society met in Jackson recently and elected the following officers for 1932: Dr. Julius Crisler, Jackson, President; Dr. T. J. Crofford, Hinds County, Dr. R. W. Smith, Madison County, Dr. W. H. Watson, Rankin County, Dr. A. E. Kennedy, Simpson County, Dr. O. O. Austin, Scott County, Dr. J. T. Rainer, Yazoo County, Vice-Presidents; and Dr. Robin Harris, Jackson, Secretary.

Claiborne County Medical Society has elected the following officers for 1932: Dr. J. V. May, Port Gibson, President; Dr. D. M. Segrest, Port Gibson, Vice-President; and Dr. G. W. Acker, Port Gibson, Secretary-Treasurer.

DeSoto County Medical Society has elected the following officers for 1932: Dr. Charles Whitley Emerson, Hernando, President; Dr. Alex J. Weissinger, Hernando, Vice-President; and Dr. L. L. Minor, Secretary-Treasurer.

The East Mississippi Medical Society has elected the following officers for 1932: Dr. J. S. Hickman, Philadelphia, President; Dr. G. L. Arrington, Lauderdale County, Dr. M. L. Flynt, Newton County, Dr. W. R. Hand, Neshoba County, Dr. T. F. Kilpatrick, Winston County, Vice-Presidents; and Dr. T. L. Bennett, Meridian, Secretary.

The Northeast Mississippi Thirteen County Medical Society, at a recent meeting, elected Dr. W. C. Spencer, Tupelo, President for 1932, and reelected all of the other officers for another year.

The North Mississippi Medical Society has elected the following officers for 1932: Dr. H. P. Boswell, New Albany, President; Dr. Frank Ferrell, Ashland, Benton County, Dr. E. S. Bramlett, Oxford, Lafayette County, Dr. C. R. Senter, Byhalia, Marshall County, Dr. W. C. Lester, Batesville, Panola County, Dr. R. M. Adams, Ripley, Tippah County, Dr. C. M. Speck, New Albany, Union County, Dr. D. C. French, Water Valley, Yalobusha County, Vice-Presidents; and Dr. A. H. Little, Oxford, Secretary.

The Jackson County Hospital staff has elected the following officers for 1932: Dr. J. N. Rape, Moss Point, President; Dr. S. B. McIlwain, Pascagoula, Vice-President; and Dr. R. G. Lander, Pascagoula, Secretary.

Dr. R. B. Yates has resumed his practice in Greenwood after an absence of some time.

Dr. D. M. Segrest, Port Gibson, has located in the office of his late father, Dr. R. A. Segrest, for the practice of medicine.

DEATHS

Dr. Thomas A. Heath, Shiloh, aged 77, died January 17 of pneumonia.

Continued on page 36

Any baby taking its daily ration of DRYCO *is automatically protected against rickets!*

"Clinical study reveals a high antirachitic potency. DRYCO produces definite healing in cases of moderate infantile rickets." (Report of Committee on Foods of the American Medical Association, J.A.M.A., Vol. 98, No. 1, p. 49., January 2, 1932.)

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New York, N. Y.

ALL DRYCO IN THE HANDS OF DRUGGISTS IS IRRADIATED

Continued from page 34

Dr. James Shamburger, Toomsuba, aged 76, died January 1.

Dr. Robert E. Higdon, Brookhaven, aged 59, died January 24.

Dr. D. P. Butler, McCall Creek, aged 61, died January 13 as the result of injuries received in an automobile accident.

Dr. E. C. Lucas, Ebenezer, aged 71, died January 9.

MISSOURI

Buchanan County Medical Society has elected the following officers for 1932: Dr. A. E. Burgher, President; Dr. Louis C. Bauman, Vice-President; Dr. Emmett F. Cook, Secretary; and Dr. J. M. Bell, reelected Treasurer; all of St. Joseph.

Cass County Medical Society has elected the following officers for 1932: Dr. B. O. Hartwell, Drexel, President; Dr. I. N. Parrish, Freeman, First Vice-President; Dr. William Beckman, Strasburg, Second Vice-President; and Dr. L. V. Murray, Pleasant Hill, reelected Secretary.

Clay County Medical Society has elected the following officers for 1932: Dr. S. D. Henry, Excelsior Springs, President; Dr. R. E. Sevier, Liberty, Vice-President; and Dr. J. J. Gaines, Excelsior Springs, reelected Secretary-Treasurer.

Franklin County Medical Society has elected the following officers for 1932: Dr. James P. Dunigan, Sullivan, President; Dr. Walter P. Mattox, Sullivan, Vice-President; and Dr. H. A. May, Washington, reelected Secretary-Treasurer.

Grundy County Medical Society has elected the following officers for 1932, Dr. W. A. Fuson, Trenton, President; Dr. W. H. Winningham, Trenton, Vice-President; and Dr. E. A. Duffy, Trenton, reelected Secretary-Treasurer.

Jefferson County Medical Society reelected all of its officers to serve for 1932 as follows: Dr. N. W. Jarvis, Festus, President; Dr. John Rutledge, Crystal City, Vice-President; and Dr. Charles E. Fallet, DeSoto, Secretary.

Johnson County Medical Society has elected the following officers for 1932: Dr. Ralph F. McKinney, Warrensburg, President; Dr. Harry F. Parker, Warrensburg, Vice-President; and Dr. O. B. Hall, Warrensburg, Secretary-Treasurer.

Lafayette County Medical Society has elected the following officers for 1932: Dr. T. R. Butler, Lexington, President; and Dr. W. E. Koppenbrink, Higginsville, reelected Secretary-Treasurer.

Nodaway County Medical Society has elected the following officers for 1932: Dr. W. M. Hindman, Burlington Junction, President; Dr. R. C. Person, Maryville, Vice-President; and Dr. Charles D. Humberd, Barnard, reelected Secretary-Treasurer.

Pettis County Medical Society has elected the following officers for 1932: Dr. A. J. Campbell, Sedalia, President; Dr. C. B. Trader, Sedalia, Vice-President; Dr. W. G. Jones, Sedalia, Secretary; and Dr. A. E. Moore, Sedalia, reelected Treasurer.

Platte County Medical Society has elected the following officers for 1932: Dr. Robert P. C. Wilson, President; and Dr. Spence Redman, reelected Secretary-Treasurer; both of Platte City.

Ste. Genevieve County Medical Society reelected all of its officers to serve for 1932 as follows: Dr. J. A. Wilkins, St. Marys, President; Dr. G. M. Rutledge, Ste. Genevieve, Vice-President; and Dr. R. W. Lanning, Ste. Genevieve, Secretary-Treasurer.

The State Board of Health has elected the following officers for 1932: Dr. Horace W. Carle, St. Joseph, President; Dr. E. Sanborn Smith, Kirksville, Vice-President; and Dr. James Stewart, Jefferson, Secretary and Health Commissioner.

A symposium on children's behavior was conducted recently at the John Burroughs School, St. Louis.

Dr. Richard Sutton, Jr., Kansas City, is spending several months at the University of Edinburgh.

Dr. John F. Hardesty, St. Louis, has been reelected Medical Director of the St. Louis Society for the Blind.

The Kansas City Southwest Clinical Society held its February clinic at St. Luke's Hospital, Kansas City, with Dr. Wilber E. Post, Chicago, as the guest speaker.

Dr. C. P. Hampton, University City, has been appointed Health Commissioner, succeeding Dr. Leo P. Fitzgerald, resigned.

St. Louis urologists recently formed the St. Louis Urological Society, and elected the following officers: Dr. Bransford Lewis, President; Dr. J. Hoy Sanford, Vice-President; and Dr. Grayson Carroll, Secretary-Treasurer.

Dr. Howard A. Dunaway and Miss Wanda Janice Saville, both of St. Louis, were married in December.

Dr. Elven Ross Cooper and Miss Olga Novelly, both of St. Louis, were married in October.

Dr. Harry J. Stein and Miss Margaret Naughton, both of St. Louis, were married January 18.

Dr. Arnold Klein, St. Louis, and Miss Ruth Birnbaum were married in October.

DEATHS

Dr. Baylor M. Spotts, Marshall, aged 65, died October 18.

Dr. Herbert C. Powers, Joplin, aged 51, died December 16 of angina pectoris.

Dr. George A. Krebs, St. Louis, aged 63, died December 5.

Dr. Max I. Birenbaum, St. Louis, aged 68, died December 14 of pneumonia.

Dr. Francis O. Drake, St. Louis, aged 91, died December 2.

Dr. James Shelton Eldredge, Kansas City, aged 61, died December 19.

Dr. Charles H. Graves, Center, aged 68, died December 18 of cerebral hemorrhage.

Dr. William F. S. Taylor, Poplar Bluff, aged 71, died January 2 of cerebral hemorrhage.

Dr. Phineas Skinner, St. Joseph, aged 59, died suddenly December 22.

Dr. Edgar William Ammann, St. Louis, aged 51, died November 24 of heart disease.

NORTH CAROLINA

Dr. W. T. Shaver, Albemarle, has been elected President of the Seventh District Medical Society.

The Eighth District Medical Society has elected the following officers: Dr. S. G. Jett, Reidsville, President; Dr. C. V. Tyner,

Continued on page 38

In Infant Feeding

Our finest testimonials are the thousands upon thousands of children, raised upon Horlick's, who, grown up, are now feeding the second and third generations upon it.

Horlick's Malted Milk is a reliable and convenient form in which to use modified cow's milk when artificial feeding is necessary.

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Klim is packed in vacuum sealed tins in 1, 2½ and 5 lb. sizes. Purchase of the larger sizes effects additional savings.

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Continued from page 36

Leaksville, Vice-President; and Dr. T. C. Redfern, Winston-Salem, Secretary-Treasurer.

Dr. J. L. Sowers, Lexington, has been elected President of the Ninth District Medical Society.

Dr. J. L. Reeves, Canton, has been elected President of the Tenth District Medical Society, and Dr. A. C. McCall, Asheville, has been reelected Secretary-Treasurer.

Salisbury physicians have formed a Medical Foundation for the purpose of erecting an office building and hospital combined, with the following serving as officers: Dr. A. S. Jones, President; Dr. J. F. Hurley, First Vice-President; Dr. J. E. Stokes, Second Vice-President; Mrs. Lyman Cotton, Third Vice-President; and Dr. H. H. Newman, Secretary-Treasurer.

Rutherford County Medical Society has elected the following officers for 1932: Dr. G. O. Moss, Cliffside, President; Dr. C. B. Buchanan, Union Mills, Vice-President; and Dr. W. C. Bostic, Forest City, Secretary-Treasurer.

Dr. Albert Anderson, Raleigh, has been elected President of the Raleigh Academy of Medicine.

Dr. Robert R. King has located in Ellenboro for the practice of medicine.

Dr. Charles A. Peterson has resumed his practice at Spruce Pine, after doing postgraduate work in New York.

The North Carolina, South Carolina and Virginia Section of the American College of Surgeons held a meeting recently at Greensboro.

The library of the late Dr. Charles O'Hagan Laughinghouse, State Health Officer, was formally presented to the State Board of Health in January, having been bequeathed to the Board of Health by Dr. Laughinghouse.

Dr. Henry Clay Salmons, Elkin, and Miss Willie Gertrude Michael were married December 17.

Dr. Edwin Jennings Cathell, Lexington, and Miss Margaret Selman were married December 26.

Dr. Rez D. Patterson, Jr., Liberty, and Miss Cleo York were married December 19.

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1. Professor Schilling's Text-Book—price $10.00
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ST. LOUIS, MO.

Dr. Lloyd R. Shaw, Statesville, and Miss Enola Gertrude Watts were married October 17.

Dr. Albertus Ula Stroupe, Jr., Mount Holly, and Miss Dorothy Lincoln were married December 31.

Dr. Wesley O. Brandon, Yadkinville, and Miss Kathleen Cooley Craver were married December 13.

Dr. Robert Boyd Rodman and Miss Elizabeth Martin Willard, both of Wilmington, were married December 31.

Dr. Martin A. Hatcher, Hamlet, and Miss Nora Langston were married December 26.

Dr. Kermit English Brown, Asheville, and Miss Rosalind Read were married December 29.

DEATHS

Dr. William Cannady Strudwick, Durham, aged 48, died January 2 of cardiorenal disease.

Dr. Edward Randolph Michaux, Greensboro, aged 72, died December 2.

Dr. Robert W. Palmer, Gulf, aged 69, died January 14 of heart disease.

Dr. William C. Brownson, Asheville, aged 75, died January 11.

OKLAHOMA

Caddo County Medical Society has elected the following officers for 1932: Dr. Wade H. Van, Cement, President; Dr. Charles R. Hume, Anadarko, Vice-President; and Dr. P. H. Anderson, Anadarko, Secretary-Treasurer.

Cleveland County Medical Society has elected the following officers for 1932: Dr. B. H. Cooley, Norman, President; Dr. William A. Meyers, Oklahoma City, Vice-President; and Dr. D. G. Willard, Norman, Secretary-Treasurer.

Creek County Medical Society has elected the following officers for 1932: Dr. J. M. Wells, Bristow, President; Dr. W. J. Neale, Drumright, Vice-President; and Dr. E. W. King, Bristow, Secretary-Treasurer.

Greer County Medical Society has elected the following officers for 1932: Dr. G. P. Cherry, President; and Dr. J. B. Hollis, Secretary; both of Mangum.

Jackson County Medical Society has elected the following officers for 1932: Dr. E. A. Crow, Olustee, reelected President; Dr. Jesse Bird, Eldorado, Vice-President; and Dr. E. W. Mabry, Altus, reelected Secretary-Treasurer.

Kay County Medical Society has elected the following officers for 1932: Dr. R. B. Gibson, Ponca City, President; Dr. L. H. Becker, Blackwell, Vice-President; and Dr. L. G. Neal, Ponca City, Secretary-Treasurer.

Kiowa County Medical Society has elected the following officers for 1932: Dr. J. D. Winter, Hobart, President; Dr. C. R. Preston, Mountain Park, Vice-President; and Dr. B. H. Watkins, Hobart, Secretary-Treasurer.

Lincoln County Medical Society has elected the following officers for 1932: Dr. A. M. Marshall, Chandler, President; Dr. F. C. Brown, Sparks, Vice-President; and Dr. J. M. Hancock, Chandler, Secretary-Treasurer.

Dr. E. H. Coachman, Muskogee, has succeeded Dr. Gene S. Atkinson as Health Officer for Muskogee County.

T. J. McGinty has resigned as Superintendent of the Oklahoma Baptist Hospital at Muskogee to take a similar position in the Davis Hospital, Pine Bluff, Arkansas.

Julia A. Milne is the newly appointed Superintendent of Sapulpa Hospital, at Sapulpa.

The Oklahoma City Clinical Society has elected the following officers for 1932: Dr. Ray M. Balyeat, President; Dr. Philip M. McNeill, Vice-President; Dr. Henry H. Turner, Director of Clinics; Dr. J. C. MacDonald, Secretary; and Dr. Earl D. McBride, Treasurer.

DEATHS

Dr. Millard Filmore Patterson, Grandfield, died December 9.

Dr. Thomas F. Wood, Sallisaw, aged 66, died December 21 following an operation.

Dr. A. C. White, Chickasha, aged 62, died January 18.

Dr. Elmer Clarence Byram, Okmulgee, aged 49, died January 23.

Dr. E. M. Wright, Olney, aged 73, died January 8.

Continued on page 40

When professional heads get together...

ALLONAL

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is invariably selected instead of the older and more highly toxic hypnotics—barbital and phenobarbital

Discriminating physicians who weigh the facts have long ago abandoned the erroneous idea that all barbituric acid hypnotics are the same in therapeutic value. Certainly there is ample pharmacological and clinical evidence to prove definitely the superiority of Allonal over remedies of its type, of barbital or phenobarbital origin . . . . .

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Issued in vials of 12 and 50 oral tablets . . . . .

A complimentary trial supply sent to physicians on request

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Continued from page 38

SOUTH CAROLINA

The South Carolina State Medical Association will hold its annual meeting in Columbia April 19, 20, and 21.

The following physicians have been elected President of their respective County Societies: Dr. D. L. Maguire, Charleston, Charleston County; Dr. Hugh Smith, Greenville, Greenville County; Dr. H. W. Corbett, Anderson, Anderson County; Dr. J. R. Allison, Columbia, Columbia County; Dr. J. M. Kibler, Newberry, Newberry County; Dr. C. P. Osteen, Sumter, Sumter County; Dr. J. T. Davis, Walhalla, Oconee County; and Dr. W. M. Sheridan, Spartanburg, Spartanburg County.

Dr. Reginald C. Alverson, Spartanburg, and Miss Ida Lee Kizer were married December 20.

Deaths

Dr. Samuel D. Parsons, Woodruff, aged 75, died December 1.

Dr. William H. Johnson, Charleston, aged 66, died December 4 of uremia.

Dr. Benjamin Broadus Steedly, Spartanburg, aged 57, died January 12 of cerebral hemorrhage.

TENNESSEE

The Tennessee State Medical Association will hold its annual meeting in Memphis April 12, 13, and 14.

Bedford County Medical Society has elected the following officers for 1932: Dr. T. J. Coble, Shelbyville, President; Dr. J D. McCord, Lynchburg, Vice-President; and Dr. W. H. Avery, Shelbyville, Secretary-Treasurer.

Blount County Medical Society has elected the following officers for 1932: Dr. D. R. Thomas, Calderwood, President; Dr. J. M. Waters, Walland, Vice-President; and Dr. J. A. McCulloch, Maryville, Secretary-Treasurer.

Campbell County Medical Society has elected the following officers for 1932: Dr. S. D. Queener, Jacksboro, President; Dr. H. T. Fortner, Jellico, Vice-President; and Dr. F. A. McClintock, Newcomb, Secretary-Treasurer.

Carter County Medical Society has elected the following officers for 1932: Dr. W. H. Longmire, Elizabethton, President; Dr. E. L. Caudill, Elizabethton, Vice-President; and Dr. E. T. Pearson, Elizabethton, Secretary-Treasurer.

Chester, Henderson and Decatur Counties Medical Society has elected the following officers for 1932: Dr. C. A. Brandon, Lexington, President; Dr. J. L. McMillen, Decaturville, and Dr. H. T. Pitts, Henderson, Vice-Presidents; and Dr. L. C. Smith, Henderson, Secretary-Treasurer.

Cocke County Medical Society has elected the following officers for 1932: Dr. M. S. Doak, Newport, President; Dr. C. W. Larue, Parrottsville, Vice-President; and Dr. J. E. Hampton, Newport, Secretary-Treasurer.

Coffee County Medical Society has elected the following officers for 1932: Dr. E. L. Womack, Summittville, President; Dr. U. B. Bowden, Pelham, Vice-President; and Dr. E. P. Vaughan, Manchester, Secretary-Treasurer.

Davidson County Medical Society has elected the following officers for 1932: Dr. C. M. Hamilton, Nashville, President; Dr. L. C. Caldwell, Nashville, Vice-President; and Dr. N. S. Shofner, Nashville, Secretary-Treasurer.

Dyer, Lake and Crockett Counties Medical Society has elected the following officers for 1932: Dr. R. W. Griffin, Tiptonville, President; Dr. W. S. Alexander, Ridgely, First Vice-President; Dr. J. L. Powell, Friendship, Second Vice-President; and Dr. Percy A. Conyers, Dyersburg, Secretary-Treasurer.

Greene County Medical Society has elected the following officers for 1932: Dr. Cecil Laughlin, Greeneville, President; Dr. R. D. Keller, Greeneville, Vice-President; and Dr. M. D. Blanton, Mosheim, Secretary-Treasurer.

Hamblen County Medical Society has elected the following officers for 1932: Dr. J. F. Campbell, President; Dr. F. F. Painter, Vice-President; and Dr. P. L. Henderson, Secretary-Treasurer; all of Morristown.

Hamilton County Medical Society has elected the following officers for 1932: Dr. B. L. Jacobs, President; Dr. H. A. Laws, Vice-President; and Dr. A. M. Patterson, Secretary-Treasurer; all of Chattanooga.

Hardin, Lawrence, Lewis, Perry and Wayne Counties Medical Society has elected the following officers for 1932: Dr. O. H. Williams, Savannah, President; and Dr. C. C. Stockard, Lawrenceburg, Secretary-Treasurer.

The Mid-South Post Graduate Medical Assembly held its annual meeting in Memphis, February 9, 10, 11, and 12.

Dr. Percy Walthall Toombs, Memphis, has been elected President of the Memphis and Shelby County Medical Society, and Dr. Jerome Logan Morgan, Memphis, Vice-President.

Dr. A. Richard Bliss, Jr., Memphis, has been elected President of the Tennessee Academy of Science.

Vanderbilt University School of Medicine has recently received two appropriations totaling three hundred thousand dollars.

Dr. Thomas M. Roberts, Sweetwater, and Mrs. Anna Belle Owens were married in October.

Dr. Robert B. Watson, Memphis, and Miss Elena Copenhaver were married in November.

Deaths

Dr. Joseph Rogers Walker, Rogersville, aged 100, died December 18.

Dr. Robert DeWitt MacDonald, Knoxville, aged 42, died December 14 in an automobile accident.

Dr. Hugh Bascom Hughes, Hampshire, aged 77, died December 19 of cerebral hemorrhage.

Dr. John J. Abernathy, Acton, aged 58, died December 31.

Dr. William Lee Abel, Dayton, aged 62, died December 18 of pulmonary tuberculosis and ulcer of the larynx.

Dr. Harry Laurin Lott, Knoxville, aged 64, died December 14.

TEXAS

The Dallas Southern Clinical Society will hold its fourth annual spring clinic conference at Dallas the week of March 28-April 2, the Baker Hotel being general headquarters. The purpose of the Society is "to make available to the medical profession of the South the postgraduate teaching material of Dallas." Dr. J. Shirley Sweeney is President; Dr. Paul H. Duff, Vice-President; Dr. Milford O. Rouse, Secretary; Dr. G. E. Brereton, Treasurer; Dr. J. L. Goforth, Director of Clinics; Dr. Curtice Rosser, Assistant Director of Clinics; and Dr. J. M. Martin, Chairman of the Executive Committee, all of Dallas.

Childress-Collingsworth-Donley-Hall-Wheeler Counties Medical Society has elected the following officers for 1932: Dr. O. L. Jenkins, Clarendon, President; Dr. F. A. White, Childress, Dr. E. W. Jones, Willington, Dr. B. L. Jenkins, Clarendon, Dr. R. E. Clark, Memphis, Vice-Presidents; and Dr. P. R. Jeter, Childress, Secretary.

Continued on page 42

Classified Advertisements

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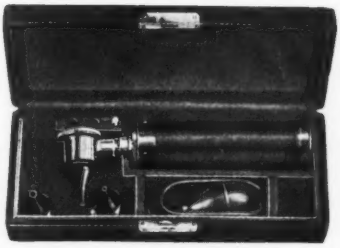

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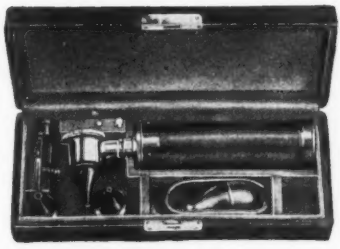

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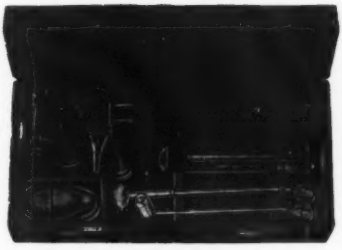

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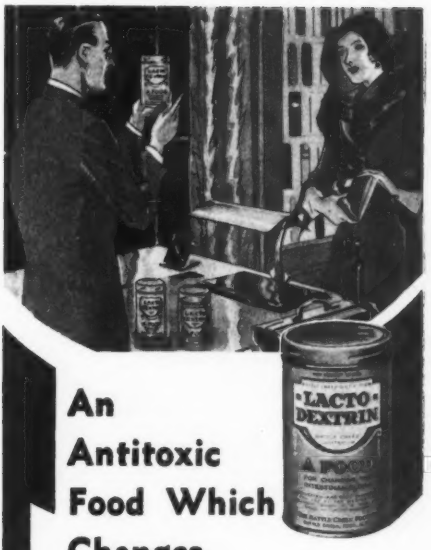

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Continued from page 40

Coleman County Medical Society has elected the following officers for 1932: Dr. E. D. McDonald, Santa Anna, President; and Dr. Maurice G. Barnes, Coleman, Secretary.

Falls County Medical Society has elected the following officers for 1932: Dr. T. G. Glass, President; Dr. S. A. Watts, Vice-President; and Dr. H. E. Hipps, Secretary; all of Marlin.

Kaufman County Medical Society has elected the following officers for 1932: Dr. J. W. Scarbrough, Terrell, President; Dr. R. J. Rowe, Kaufman, Vice-President; and Dr. D. H. Hudgins, Forney, Secretary-Treasurer.

McCulloch County Medical Society has elected the following officers for 1932: Dr. J. B. Granville, Brady, President; Dr. J. E. Powell, Brady, Vice-President; and Dr. Oscar Huff, Mason, Secretary-Treasurer.

Medina - Uvalde - Maverick-Val Verde-Terrell-Edwards-Real-McKinney-Zavala Counties Medical Society has elected the following officers for 1932: Dr. R. A. Eads, Uvalde, President; Dr. W. P. Meredith, Del Rio, Vice-President; and Dr. Cary Poindexter, Crystal City, Secretary-Treasurer.

Nacogdoches County Medical Society has elected the following officers for 1932: Dr. T. J. Blackwell, President; and Dr. Henry Tucker, Secretary-Treasurer; both of Nacogdoches.

Navarro County Medical Society has elected the following officers for 1932: Dr. T. O. Wills, Corsicana, President; Dr. Dubart Miller, Corsicana, Vice-President; and Dr. William T. Shell, Jr., Corsicana, reelected Secretary-Treasurer.

Nolan County Medical Society has elected the following officers for 1932: Dr. Thomas Slayden, President; Dr. A. A. Chapman, Vice-President; and Dr. R. O. Peters, Secretary-Treasurer; all of Sweetwater.

Nueces County Medical Society has elected the following officers for 1932: Dr. N. D. Carter, President; Dr. McIver Furman, Vice-President; and Dr. Cedric Priday, Secretary-Treasurer; all of Corpus Christi.

Potter County Medical Society has elected the following officers for 1932: Dr. J. J. Crume, President; Dr. W. J. Shudde, Vice-President; and Dr. R. R. Swindell, Secretary-Treasurer; all of Amarillo.

Tom Greene County Medical Society has elected the following officers for 1932: Dr. J. B. Chaffin, President; Dr. S. J. Burleson, Vice-President; Dr. L. O. Woodward, Secretary; and Dr. H. R. Wardlaw, reelected Treasurer; all of San Angelo.

Van Zandt County Medical Society has elected the following officers for 1932: Dr. Ben B. Brandon, Edgewood, President; Dr. Felix V. Bryant, Martin's Mill, Vice-President; and Dr. D. Leon Sanders, Wills Point, reelected Secretary-Treasurer.

Webb County Medical Society has elected the following officers for 1932: Dr. A. T. Cook, Laredo, President; Dr. Thomas Stetson, Hebbronville, Vice-President; and Dr. Raul de la Garza, Laredo, Secretary-Treasurer.

Williamson County Medical Society has elected the following officers for 1932: Dr. B. A. Kirkpatrick, Thrall, President; Dr. C. C. Foster, Granger, Vice-President; and Dr. Van C. Tipton, Georgetown, Secretary-Treasurer.

Dr. Ray K. Daily, Houston, has been elected President of the Staff of the Baptist Hospital at Houston.

Dr. Max R. Woodward, Sherman, is in Vienna, Austria, doing postgraduate work.

Dr. Lois Wier Smith has become associated with the Stout Clinic of Sherman, moving to Sherman from Dallas.

The State Tuberculosis Sanatorium, San Angelo, is building an addition to its present dormitories, which will have a 60-bed capacity.

San Angelo and West Texas will have a memorial hospital erected from a million and a half dollar bequest for that purpose.

The Texas Public Health Association met in Houston in November for its ninth annual session with a registered attendance of over 200.

The Texas Neurological Society met in Austin in November for its semi-annual session.

Dr. J. W. Bass, Dallas, has been appointed Director of Public Health of the City of Dallas.

Dr. John F. Jubben, Jr., Dallas, and Miss Marie Elizabeth Mahoney were married in New York November 29.

The Baptist Hospital, Houston, is now known as Memorial Hospital, this change having been made January 1.

Deaths

Dr. Dru McMickin, Beaumont, aged 61, died suddenly November 26.

Dr. William Davis Northcutt, Longview, aged 70, died November 20.

Dr. John E. Copenhaver, Pilot Point, aged 65, died December 8 of cerebral hemorrhage.

Dr. Julius Zulch, Zulch, aged 62, died November 21 of pneumonia.

Dr. Edgar Bacon Stokes, Crockett, aged 65, died December 9.

Dr. Larkin B. Miller, Dallas, aged 63, died October 19.

Dr. Charles Binney, Strawn, aged 68, died December 7.

Dr. Frank Dennis Shepherd, Wichita Falls, aged 67, died October 18.

Dr. Charles Miller Hatcher, Presidio, aged 43, died December 27.

Dr. J. P. Worsham, Barry, aged 60, died November 13 following an operation.

Dr. W. P. Irvine, Mabank, aged 69, died October 6.

VIRGINIA

The James City-New Kent Medical Society, at a recent meeting, reelected the same officers for 1932, as follows: Dr. A. M. Sneed, Toano, President; Dr. J. R. Parker, Providence Forge, Vice-President; and Dr. J. R. Tucker, Williamsburg, Secretary-Treasurer.

Halifax County Medical Society has elected the following officers for 1932: Dr. C. B. White, Halifax, President; Dr. George A. Stover, South Boston, Vice-President; and Dr. William C. Brann, South Boston, Secretary-Treasurer.

Continued on page 44

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Continued from page 42

Loudoun County Medical Society has elected the following officers for 1932: Dr. M. H. Hiden, Warrenton, President; and Dr. John A. Gibson, Leesburg, Secretary.

The following have been elected officers of the Petersburg Hospital Staff: Dr. Herbert C. Jones, Chairman; Dr. E. W. Young, Vice-Chairman; Dr. Leta J. White, Secretary; Dr. H. M. Snead, Vice-Secretary; and Dr. George H. Reese, member of medical committee.

The Roanoke Academy of Medicine has elected the following officers for 1932: Dr. Everett E. Watson, Salem, President; Dr. F. A. Farmer and Dr. E. C. Ambler, both of Roanoke, Vice-Presidents; and Dr. Charles H. Peterson, Roanoke, Secretary-Treasurer.

Dr. D. Lane Elder, Hopewell, has been elected District Trustee of the Kiwanis Club of Hopewell for 1932.

Dr. Harvey B. Haag, Richmond, has returned to his duties at the Medical College of Virginia after six months' study in Germany.

Dr. George H. Snead, Richmond, has sailed for Europe for several months of study on the Continent.

Dr. Stuart McGuire, Richmond, has been appointed a member of the Richmond Public Library Board.

Dr. Oscar L. Hite has opened offices at Richmond for the practice of gastroenterology.

The Richmond Ophthalmological and Otolaryngological Society at a recent meeting elected the following officers for 1932: Dr. W. R. Weisiger, President; Dr. T. E. Hughes, Vice-President; and Dr. R. W. Vaughan, Secretary-Treasurer.

Dr. John A. Pilcher, Jr., has located in Roanoke to practice, and is a member of the Staff of the Gill Memorial Eye, Ear and Throat Hospital.

Dr. John Braxton McKee, Winchester, and Miss Ruth Loraine DeBussey were married January 1.

Dr. James Alfred Gooch and Miss Lelia Watkins Stacy, both of Richmond, were married January 4.

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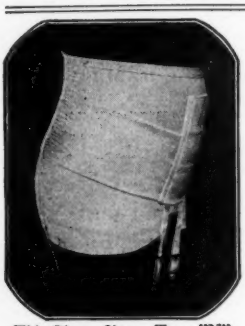

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Deaths

Dr. Edward Thomas Hargrave, Norfolk, aged 60, died December 1.

Dr. Giles Burneston Cook, Front Royal, aged 54, died recently in a hospital in Washington, D. C.

Dr. Lorimer Fauntleroy James, Richmond, aged 58, died December 28 of heart disease.

WEST VIRGINIA

Dr. James R. Bloss, Huntington, has been appointed the member of the Council from West Virginia of the Southern Medical Association, the appointment having been announced recently by the President, Dr. Lewis J. Moorman, Oklahoma City, Oklahoma. Dr. Bloss succeeds Dr. Walter E. Vest, of Huntington, whose term had expired, and who having served the constitutional limit, was not eligible to reappointment.

Cabell County Medical Society has elected the following officers for 1932: Dr. Raymond M. Sloan, Huntington, President; Dr. W. Byrd Hunter, Huntington, Vice-President; Dr. William W. Strange, Huntington, Secretary; and Dr. Ray M. Bobbitt, Huntington, reelected Treasurer.

Eastern Panhandle Medical Association has elected the following officers for 1932: Dr. R. B. Talbott, Martinsburg, President; Dr. J. J. Pittman, Charles Town, Vice-President; and Dr. Edwin Cameron, Martinsburg, Secretary-Treasurer.

Grant-Hardy-Hampshire-Mineral County Medical Society has elected the following officers for 1932: Dr. Glenn Moomau, Petersburg, President; Dr. J. B. Grove, Petersburg, Dr. Harry Rawlings, Jr., Wardensville, Dr. James Daily, Romney, Dr. James T. Wolverton, Piedmont, and Dr. S. B. Johnson, Keyser, Vice-Presidents; and Dr. T. C. Giffin, Keyser, reelected Secretary-Treasurer.

Greenbrier Valley Medical Society has elected the following officers for 1932: Dr. H. C. Solter, Marlinton, President; Dr. W. E. Myles, White Sulphur Springs, reelected Vice-President; and Dr. N. R. Price, Marlinton, Secretary-Treasurer.

Kanawha Medical Society has elected the following officers for 1932: Dr. W. P. Black, President; Dr. B. S. Preston, Vice-President; and Dr. M. F. Peterson, Secretary-Treasurer; all of Charleston.

Logan County Medical Society has elected the following officers for 1932: Dr. H. H. Farley, Logan, President; Dr. B. D. Smith, Omar, Vice-President; and Dr. Fred E. Brammer, Dehue, Secretary-Treasurer.

Marion County Medical Society has elected the following officers for 1932: Dr. Grady Morgan, Fairmont, President; Dr. D. D. Hamilton, Mannington, Vice-President; Dr. Charles T. Francis, Fairmont, Secretary; and Dr. C. W. Waddell, Fairmont, Treasurer.

Marshall County Medical Society has elected the following officers for 1932: Dr. O. P. Wilson, Moundsville, President; Dr. S. F. Yoho, Francis, Vice-President; Dr. J. A. Striebich, Moundsville, reelected Secretary; and Dr. L. H. McCuskey, Moundsville, Treasurer.

Mingo County Medical Society has elected the following officers for 1932: Dr. L. S. Henley, Borderland, President; Dr. G. W. Easley, Williamson, Vice-President; and Dr. Fred Ben Quincy, Williamson, Secretary-Treasurer.

Wyoming County Medical Society has elected the following officers for 1932: Dr. J. F. Biggart, President; Dr. B. W. Steele, Vice-President; and Dr. Ward Wylie, Secretary-Treasurer; all of Mullens.

Dr. John Breckinridge Woodville, Jr., Lansing, and Miss Roberta Lee Walters were married January 13.

Deaths

Dr. Curtis Taploff Arnett, Clarksburg, aged 61, died December 5.

Dr. Irvin B. Keller, Jane Lew, aged 55, died October 12 of angina pectoris.

Dr. John Eldon Corbin, Clarksburg, aged 51, died December 16 of influenza.

Dr. Ernest R. Logan, Omar, aged 41, died January 13 of pneumonia.

Dr. Elmer Perry, Fort Spring, aged 63, died January 7.

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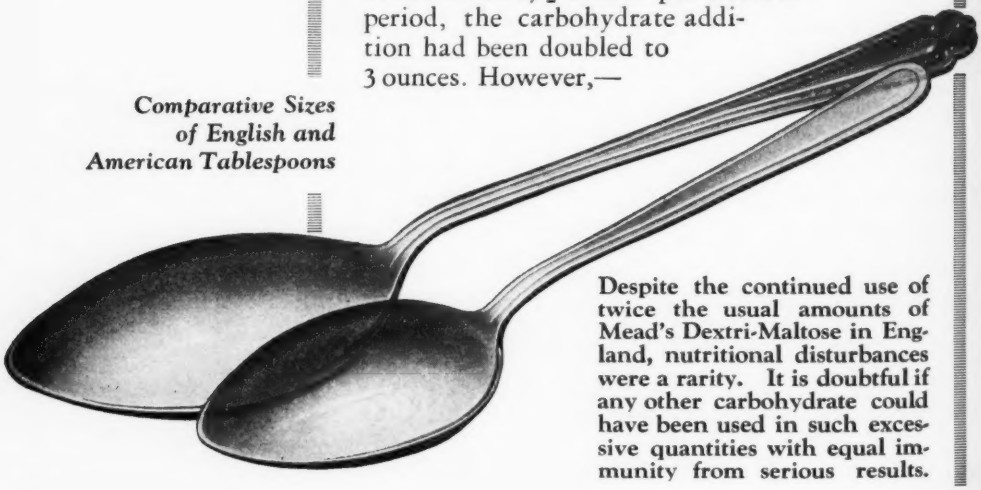
Comparative Sizes of English and American Tablespoons



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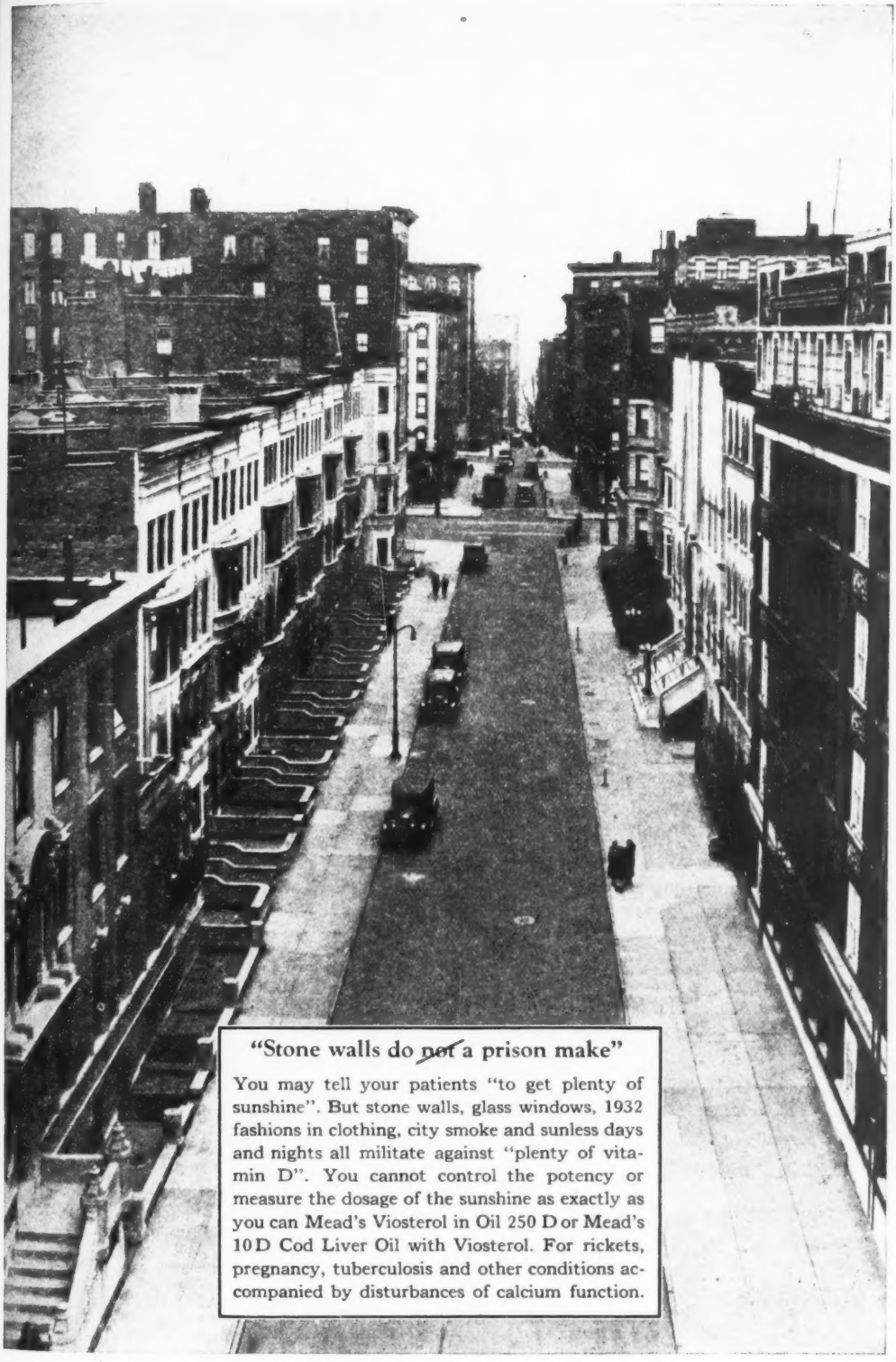

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